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APHASIA
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APHASIA IN CHILDREN

by

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With an Introduction by

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INTRODUCTION

So much has been written on the subject of Aphasia that perhaps some kind of introduction is needed to justify the present volume. If so, it certainly deserves to be written by some one with a much greater experience of Aphasia than I have, but fortunately all that needs to be done is to point out that Dr. Ewing's book is concerned with a type of speech defect which is due to causes entirely different from those responsible for aphasia in the adult. The aphasia which has been so much studied is due to a breakdown occurring in the complex machinery set up in infancy for 'symbolic thinking and expression'. In the children whom Dr. Ewing has investigated with so much care there has been a failure to set up the machinery in the first instance, and we are invited to consider the conditions which are essential for the development of the speech mechanism and not the result of its disintegration from brain injury in later years.

His analysis turns on the discovery of an extremely interesting state of partial deafness which deprives the spoken word of all its meaning by making the higher vibration frequencies inaudible. The child can hear something, but speech must sound to him like the mumblings of a loud speaker on a badly adjusted wireless set, with the added disadvantage that the frequencies which are inaudible are just those needed to distinguish one speech sound from another. Small wonder that the child turns an apparently deaf ear to the meaningless noises made by his parents, that he fails to develop a speech mechanism of his own, and that he can do so as soon as the appropriate training has shown that the sounds are really not indistinguishable if they are listened to attentively.

To arrive at this result Dr. Ewing has had to test the whole range of hearing in children who can neither speak nor understand speech, and he has devised an excellent way of doing it. The sections of the book which describe his method and discuss the testing of auditory acuity in general will be of great interest to otologists, as his comparison of aphasic conditions in adults and children will be to all who are concerned with the

central mechanisms of speech. But its chief claim must be that his book describes a form of speech defect in young children, the cause of which has been unrecognized in the past, and that a knowledge of the cause has made it possible to educate the patients not merely to speak but, within limits, to hear as well.

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May 1930.

EXPLANATORY NOTE

THIS book is a brief account of the methods by which it was discovered that of ten congenitally 'aphasic' boys and girls six suffered from a condition of partial deafness hitherto unknown and unstudied. Prior to this research they had been variously classed as aphasic, as totally deaf, or as having normal hearing and being totally or almost totally dumb for some cause unknown. The symptoms which led to such varying conclusions are here traced to a condition of normal or relatively normal hearing for a certain range of low tones, associated with marked deafness to high tones. This group of patients (Nos. 1–6) are therefore described as 'high-frequency deaf'.

Another patient (No. 7) previously diagnosed as born deaf and dumb proved, with the new tests, to have normal hearing.

Three more patients (Nos. 8–10) exhibited gross defects of speech but were shown to have normal hearing. In every instance the comprehension as well as the use of words was disturbed. The most obvious trouble of No. 10 was inability to read or write, but close examination revealed a general defect of speech and language.

The data in the case of the non-deaf patients (Nos. 7–10) indicate incomplete or arrested development of the central mechanism—in the brain. Yet general intelligence, apart from the use of words, proved normal.

This investigation, however, showed such radical differences between the forms of trouble found among these four patients, and those due to injuries of the brain, studied by other workers and described as 'aphasia', that a new term has been adopted here—'linguistic retardation'.

The above conclusions were only reached after the examination by the same methods of ninety other children and adults, normal and deaf.

Nos. 11–17 were deaf and normal adults.

No. 18 was an 'aphasic' boy not available during the whole period of the research.

Two adult aphasics (Nos. 19 and 20) were observed and tested.

Nos. 101–12 were normal children in a public infant and elementary school.

Nos. 201–71 were pupils in a residential school for the deaf.

My best thanks are due to all who have facilitated this investigation: particularly to Professor J. S. B. Stopford, who generously undertook to supervise it; to Dr. E. D. Adrian and Dr. F. W. Lamb for much helpful criticism and advice; to Dr. James Drever for the use of his 'Performance Tests of Intelligence' before their publication in 1928; to Dr. R. H. Thouless for valuable suggestions; to Dr. H. C. Smith for examining the ears of patients; to Mr. D. McIntosh for assisting in the experiments with sensitive flames; most of all to my wife, but for whose constant help and rare capacity for understanding and teaching normal and abnormal children the investigation would never have been made.

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May 1930.

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CHAPTER I

THE NATURE OF THE PROBLEM

FIRST OBSERVATIONS

In March 1924 a little boy of $4\frac{1}{2}$ years was brought to me. Up to the age of 3 years he had given no sign of being able to hear. His father, who is in medical practice, took him to a consultant for an aural examination. The pupils of his eyes were found to dilate when a tuning-fork was sounded close to his head. Congenital adenoids were diagnosed and removed. There was no history of deafness in the family, the patient had not suffered from any injury or serious illness, and the condition of the ears was consistent with good hearing. However, another consultant decided from the apparent absence of response to sound that there was total deafness.

Between the ages of 3 and 4 years this boy occasionally responded to sound. If his father entered the room where he was and shouted 'David' (his name) he would turn round if not engrossed in play. But if his attention were already occupied he showed no sign of having heard any sound. A similar occasional and variable reaction was produced when a motor-horn was sounded close behind his back.

When I first saw him in March 1924—he has come to me for daily teaching by special methods and has therefore been under continuous observation ever since—he behaved on the whole like a congenitally deaf child. He could not understand a word said to him by any one. He could not speak a word. He gave no reaction to any form of noise during the first two or three weeks. He expressed his wants by means of gestures and cries which were not words. ('Mummy', when he was displeased or wanting something, might or might not be a rudimentary word—it was sometimes used when his mother was not present and may have been a purely emotional cry.)

Yet it was noticeable that in his cries there was normal intonation of the voice, not found in any but very partial conditions of congenital or pre-linguistic (see p. 11) deafness. His movements were quick, neat, and finished. Unlike the

born-deaf child he did not slur his feet when walking. A very marked feature of his behaviour, never to my knowledge observable among children suffering from congenital or early acquired deafness, was an extraordinary quickness of head and eye, often of bodily movement. His attention obviously flashed from one object to another at an unusually rapid rate. This characteristic was aptly described by a medical observer in a similar case as 'bird-wittedness'. An equally important factor was the strength of his emotional reactions. His parents and nurse told how he would have violent fits of passion and had been known to throw his clothes into the fire.

(The history of this patient, No. 1, is given in greater detail, Chaps. IV and VI.)

From time to time I had seen something of a little girl whose behaviour was precisely similar as regards all these outstanding characteristics (No. 2, Chaps. IV and VI). She was brought to Mrs. Ewing for special teaching in September 1922, at the age of $4\frac{1}{2}$ years. She was transferred to my care in May 1925 and since June 1927 has visited me daily. There was on the one hand a similar diagnosis by a consultant of total deafness, though there was no indication of any lesion; there had been no serious illness and no injury. Her regular medical attendant, however, in June 1926 declined to express an opinion as to whether she was deaf, and if so, whether the trouble was organic or functional (present condition of ears, see p. 149).

A third case was described to me in detail by Mrs. Ewing, who had her under her care for $3\frac{1}{2}$ years, from the age of 4 years 9 months to 8 years 3 months (No. 7, pp. 121–4 and 136). Again the behaviour was precisely similar as to all the characteristics described above. There was a diagnosis of total deafness of congenital origin.

All these three children, therefore, were deaf to speech before special teaching was begun. They could not speak. Their response to sound was uncertain, variable, and occasional. They were all physically well formed, without history of disease or injury. Diagnoses of their condition were contradictory. Their behaviour was distinguished from that of the young deaf child by important features, natural tone of voice, quiet movements, especially in walking, and

the marked alertness and rapidity of changes in attention described as 'bird-wittedness'. The intelligence of their reactions to situations, apart from the use of words and response to sound, was of a strikingly high order. Finally, all these three patients, Nos. 1, 2, and 7, began to react quite definitely and clearly to the sound of the human voice at periods varying from one to three months from the beginning of special teaching.

These three patients therefore presented a difficult problem. Clearly, the most effective special teaching must be based on exact knowledge of their condition. But such knowledge did not appear to be available.

METHODS OF INVESTIGATION

Since there was no evidence of ear disease to account for the gross apparent deafness it was necessary to inquire whether this was a defect of the central mechanism for speech—in the brain. Such disorders have hitherto been described as aphasia. At the same time the contradictory character of the diagnoses mentioned indicated that the 'routine' tests of hearing were ineffective in such conditions and some other means had to be found to decide whether the children were deaf or not deaf.

The whole of this research, therefore, was an attempt to answer two questions:

1. Were these patients aphasic?
2. Were these patients deaf?

The methods adopted have been: (*a*) Clinical, close observation of the patients' behaviour from day to day. This method has proved most fruitful in investigating aphasia. Jackson (p. 83) used no other.

The patient's behaviour, especially as regards speech and language, was recorded in diary form, and analysed in the light of data collected by workers on aphasia. Other children suffering from an 'aphasic' condition were found. Two adult aphasics were closely observed (while coming to me for re-education) for periods of twelve and eighteen months respectively. The whole body of data was sifted and compared at intervals. For the first time, I believe, an attempt has been made to discover whether any common factors underlie congenital and acquired (i.e. post-linguistic) aphasia.

(*b*) Experimental. It proved necessary to find new tests

with new or special apparatus to investigate hearing capacity. Certain data obtained from workers in physics and experimental psychology were discovered to have the greatest significance in the development of speech and language in the child. The new test for auditory acuity was given to 12 'normal' children in an infant and an elementary school and 71 deaf children receiving special education. This test has since been adopted by the Department for Training Teachers of the Deaf at Manchester University for testing a still larger number of children in special schools for the deaf, and the work is being carried on at the present time.

The speech and language capacity of patients Nos. 1–10 was also tested under experimental conditions.

In both the clinical and experimental work it seemed clear that the results must be checked by the reactions of normal children in similar—in the case of experimental tests identical—conditions. Previously this had been done by Burt[1] alone of all the workers quoted in Chapter V. Any other form of standardization is purely arbitrary.

DEVELOPMENT OF SPEECH AND LANGUAGE IN THE NORMAL CHILD

A rich mine of data on normal language processes has hitherto scarcely been tapped in connexion with aphasic disorders, i.e. studies of normal children, especially with regard to speech and language development. A scientific technique has been evolved and much reliable data collected by workers in this field.

Outstanding and thoroughly representative contemporary workers are, in child psychology, W. and C. Stern,[2] and Descoeudres[3] of the J. J. Rousseau Institute at Geneva; in the philology of children's speech Jespersen,[4] and in the comparative psychology of speech, de Laguna.[5]

An analysis of their data and conclusions reveals agreement on fundamental points.

Stern, de Laguna, and Jespersen agree in recognizing the following stages of speech and language development in the normal child.

[1] See p. 107. [2] See bibliography. [3] Ibid.
[4] Ibid. [5] Ibid.

A. *The pre-linguistic period, before either the comprehension or the utterance of speech proper has begun.*

At birth hearing is the least developed of the special senses and only 'shock reactions' are possible.[1] In the first half-year to screaming (initially an instinctive expression of the general condition, the feeling-tone[2]) is added the phenomenon of 'babbling'.[3] This is characterized by perseveration (repetitions such as bar, bar, bar, &c.) with natural changes in intonation. de Laguna has pointed out that in the bird and animal world the various cries fulfil a social purpose in enabling the 'synchronization of physiological states', e.g. in pigeons during the mating process.[4] Stern and Jespersen observed that the change from screaming to babbling is towards a type of utterance more utilizable for human speech.[5] Babbling, however, begins in deaf children just as among the hearing.[6] It is therefore an inherited reaction not dependent for its fructification on auditory stimulation. Some of my patients suffering from defects of speech are known not to have 'babbled', notably No. 7 of those mentioned in this book. No. 8 probably was another instance, as far as his mother is able to give me information. Nos. 1 and 2 definitely did babble. It seems likely that the symptom of not babbling is much more important than has hitherto been realized (p. 144 below).

During the second half-year, with most children a new feature, that of imitation, becomes noticeable in the babbling.[7] Previously the sounds uttered by the child have not closely resembled those in the speech that he is hearing used in his environment. Stern says that the child 'approaches speech along three lines:

(1) the active expression of childish babble,
(2) unintelligent imitations, and
(3) understanding what is said to it.'[8]

He adds: 'possibly the child has for minutes babbled "Mamam" . . . but the babble-sound becomes a babble-word, i.e. speech,

[1] Stern, 74. [2] Ibid. 125.
[3] Ibid. 141, and Jespersen, 104.
[4] de Laguna, 25, 76, and 256. [5] Stern, 141, and Jespersen, 104.
[6] Stern, 141 (Mrs. Ewing has observed this.)
[7] Stern, 141, and Jespersen, 106. [8] Stern, 142.

when he subjectively connects some meaning with mamam.' Both Stern and Jespersen emphasize the importance of what is deliberately said to the child by parents or nurse.[1]

This merges into the period of the so-called 'Little Language' of children, e.g. the boy at 18 months who called himself 'Dodado'. My patient No. 1 in the first year of special teaching called himself 'Stotter'. The child's imitations of his elders are imperfect and approximate to his own babblings.[2]

B. *The 'one-word sentence'.*

At a varying age—about the beginning of the second year—appears the 'one-word sentence'.[3] Stern emphasizes the conative aspects of its use. de Laguna illustrates this stage by the following. A French girl (aged 2 years) 'pointed with her finger to her brother's slippers . . . saying simply, "Mon-Mon" (Raymond) . . . she brought her mother's boots, and . . . said, "Maman"; she offered me the paper saying, "Papa"; she wanted to have a box of marbles belonging to her brother, and, stretching out her hand, she said, "Mon-Mon". In all these cases the object was indicated by a gesture'.[4] Jespersen denies the assertion of earlier workers that the 'one-word sentence' is a purely emotional outburst, but adds the statement—significant in view of all that has been written by the authorities on aphasia from Hughlings Jackson to Head (Chapter V)—'without strong feelings the child would not say much'.[5]

Intonation and gesture are of greater relative importance in helping the child to make his feelings and wants appreciated at this stage than in later life.[6]

About the middle of the second year of life or later comes a time when he gradually or perhaps almost suddenly realizes the meaning and purpose of speech and that everything has a name.[7] He learns 'to discriminate clearly the different features of his environment by giving the appropriate verbal response to each'.[8] Thus this is a period of general mental as

[1] Cf. Jespersen, 104. [2] Stern, 145, and Jespersen, 106.
[3] Stern, 149, de Laguna, 86–94 and 262–71, Jespersen, 133.
[4] de Laguna, 88. [5] Jespersen, 134.
[6] Stern, 149, and de Laguna, 70. [7] Stern, 162.
[8] de Laguna, 271.

well as linguistic development, the two forms of growth interacting and facilitating each other.

At every stage the development of the speech-language function has this dual aspect—the mastery of inflexions is needed for and helps towards realization of 'the finer shades of ideas',[1] compound sentences are required by and aid the increasing complexity of the child's thinking.

The 'naming' period is described by Stern as one in which vocabulary increases 'by leaps and bounds'.[2] His children, Hilde and Gunther, used 'that! that!' and 'that? that?' as a form of request to be told the name of everything.

My patient No. 1 used 'Ti-ta' in the second month or so of special teaching, later, if not at first, for the same purpose. Probably it began as an echo-like imitation and passed through an intermediate stage of being 'a social game'—accompanied by a pointing gesture, as in the instance of one of de Laguna's own children, who began to say 'ti-ti' after her grandfather when he was showing her his watch, and then used it as a means of calling attention to any object.[3]

C. *Words grouped in sentences.*

The third period begins as the child passes from the 'one-word sentence' to groupings of words. This may occur between the ages 18 months and 24 months,[4] but with some normal children it happens later. The beginnings of this period are characterized by such juxtapositions of words as 'My go snow' ('I want to go in the snow'), &c.[5] 'The child becomes capable of connecting several words to express one thought.'[6]

Stern remarks: 'these word collections were not all so fluently spoken as our sentences; their characteristic was rather a jerky utterance . . . of the isolated words.'[7] Perhaps this is partly because the child has not yet developed the hierarchies of habits necessary for such complex use of the speech mechanism—the utterance of sentences requires a most intricately controlled use of three groups of organs—respiratory, laryngeal, and articulating. Partly also it may be that the fusion of several thoughts and attitudes into one

[1] Stern, 172. [2] Ibid. 162. [3] de Laguna, 70.
[4] Stern, 163, and Jespersen, 135. [5] Jespersen, 134.
[6] Stern, 163. [7] Ibid. 164.

whole is as yet incompletely effected. Aphasic patients sometimes manifest remarkably similar phenomena (pp. 97, 103, and 137).

D. *Acquisition of inflexions.*

At the age of 2 years or 2 years 6 months children may begin to use 'the different forms of inflexion (declension, conjugation, comparison); this stage "lasts some years" and four- or five-year-old children "often have a hard struggle" with these forms.[1] Irregular forms present difficulty, e.g. "drinked" for "drunk", "badder" for "worse"'.[2] Stern illustrates this period: 'Mama—wanty pickies—room—wanty pickies—back—dada, mama fetch', indicating 'a wish that the child's mother should fetch pictures out of the back room'.[3]

The use of a plural form is noticeable. Questions are asked in this stage ('Apple where?' = 'Where is the apple?') and varieties of sentence begin to appear.[4]

E. *Compound sentences.*

The child enters upon the fourth stage of speech-language development proper when he begins to use compound sentences with subordinate clauses. Stern's daughter Hilde at 3 years said: 'I will look in the kitchen and ask if she is coming here'; 'You'll get no bread and butter if you're so naughty.'

MISTAKES IN THE SPEECH OF NORMAL CHILDREN

In the study of defects of speech among children the 'mistakes' of normal children are of special interest.

These have been closely analysed by Stern and Jespersen. Stern says: 'Every word that is to be reproduced must be (1) heard, (2) noted, (3) pronounced, and (4) remembered.'

Hence fine shades of difference in sound may not at first be noticed. Vowels are 'less often mutilated than consonants' (cf. notes on relative intensity and pitch-complexity of vowels and consonants, pp. 56–7). The gutturals and sibilants of the consonants are most often wrongly reproduced. 'Since attention generally outruns pronunciation, and is principally directed to the more prominent parts of a word, . . . stressed syllables (suffer) least. . . .

[1] Stern, 166. [2] Ibid. [3] Ibid. 166. [4] Ibid. 167.

So we meet in children's speech:

1. The elision of single sounds (o-o = cocoa; ittle = little).
2. Or whole syllables (mergence = emergency).
3. Change of sound, substitution of dentals for gutturals (dood = good; toffee-tan = coffee-can).
4. Assimilation, i.e. making one sound in a word like another (gugar = sugar; goggie = doggie; bobbin = dobbin).
5. Metathesis, i.e. transposition of sound (waps = wasp; evnelope = envelope).
6. Contamination, i.e. the combination of the elements of two words (breakolate = breakfast and chocolate).'[1]

Jespersen also gives instances of these tendencies.[2]

He notes the difficulty shown by most children in learning to use the consonant 'R', the trouble caused to a child of 2 years by 'TH'—a common phenomenon resulting in 'free' for 'three', &c. (Adult patients as well as children sometimes come to me who cannot say 'R').

Reduplication (water-water for water) was a form of perseveration observed by Jespersen among the children whose speech he recorded.

TESTS OF VOCABULARY

Descoeudres 'interrogated individually each of 300 subjects'.[3] Her results were experimental and quantitative. Her most important tests from the point of view of this research were those of 'Comprehension of language—execution of 10 easy commands, pointing out 10 actual objects, and pointing out pictures'—named by herself as observer; also tests involving repetition of figures and syllables and tests of vocabulary.[4] Her work covered the age period two years to eight years, and was chiefly carried out with 'working-class' children,[5] unlike that of most investigators in this field, who have observed children of the middle and upper classes. Her principal general conclusions were that:—

1. 'there are certain moments when the development of the child is more marked'—in the second half of the fifth year and the first half of the seventh year.[6]

[1] Stern, 155–6. [2] Jespersen, 107–22. [3] Descoeudres, 13.
[4] Ibid. 36–7. [5] Ibid. 315. [6] Ibid. 311.

2. There is no general difference in development between girls and boys, but there are differences in specific abilities—e.g. in manual skill the girls lead. Differences between individuals are more marked.[1]
3. In language the children of the well-to-do class show greater development at the various ages. She attributes this fact to their hearing a great deal of speech in their environment and to the smaller classes in the schools they attend.[2] There was, however, no difference between the children from the social classes in the repetition of syllables.[3]

The mass of data available from the work of these four (and other) investigators is indispensable to the study of aphasia in young children. With this knowledge it becomes possible to discriminate between 'errors' natural to normal development at the various stages and abnormal features in the speech and language of patients.

HEARING ESSENTIAL TO THE NATURAL DEVELOPMENT OF SPEECH AND LANGUAGE

In the speech and language development of the normal child, therefore, the essential processes are first hearing, then learning to understand and to imitate the spoken word, accurate hearing of the whole of words being an indispensable requirement.

EFFECTS OF CONGENITAL AND ACQUIRED DEAFNESS ON SPEECH

Quite apart from the evidence quoted in the present and previous chapter there are the facts of deaf-mutism. In the absence of hearing, neither understanding of words, speech, reading, nor writing develop naturally. In order that the deaf may react to words or use words, other 'ways-in' have to be found, through the eye and through touch. Ten years of strenuous special teaching, while working wonders and providing them with the essential means of communication with society through lip-reading, speech, reading, and writing, rarely succeed in giving them such command of speech and language

[1] Descoeudres, 313. [2] Ibid. 315. [3] Ibid. 316.

as would be normal at their age and in their home environment. Even acquired deafness occurring before the age of five, if at all severe, almost always destroys speech. Occurring later it frequently produces what may be described as a partial 'atrophy' of speech, even in adults: the voice sometimes becomes monotonous and pronunciation imperfect.

In individual development, therefore, understanding of speech and reactions to speech heard occur first, the utterance of words begins to develop a little later and is dependent on the auditory function for its origination and largely for its maintenance. Reading and writing also are normally acquired with the help of hearing and speaking. At first the teacher points to the written or printed word or letters and says the word indicated and the child begins, almost always, by reading aloud.

THE UNITY OF THE SPEECH AND LANGUAGE FUNCTION

This ultimate dependence of all speech and use of words, in the normal individual, on hearing, provides one possible reason for the conclusion of Hughlings Jackson, Moutier and Marie, and Head, quoted in Chapter V, that in adult aphasia, where speech is affected, reading and writing suffer also. Similarly, the observations of Burt, Fildes, and Hincks (Chap. V) that auditory acuity or auditory memory or both are often subnormal in children who show reading or writing disability, become of great significance. The present research has produced additional data on this point.

Briefly, it seems clear that hearing and understanding words, speech, reading, and writing form one closely interwoven function. In race-history reading and writing are of late development. Many living educated persons have only had a few ancestors who could read or write. Speech, on the other hand, goes back to such a remote era that its origin provides a never-ending controversy. The human child, even if deaf, goes through a period of babbling, i.e. of incipient speech, though this cannot become true speech except through the constant stimulus of speech heard. It is thus most probable that speech has an inherited basis in the nervous system. Hearing has been found most acute over the pitch-range of human speech (p. 51). The speech organs appear to have a

long history of special development quite apart from their use for other purposes, e.g. of the tongue in eating.

Lack of hearing therefore is a constant factor capable of either impairing or preventing the natural development of the speech language function. It is contrasted with defects of speech and language, such as—frequently—aphasia, where the patient may or may not be able to call a chair by its proper name on successive days. Here the defect is clearly variable, though the effectiveness of the stimulus in eliciting the appropriate reaction probably depends on factors of a conditioned reflex character, as with a stammerer who talks freely to his family but cannot make a simple purchase in a shop without infinite difficulty. With the aphasic patient there are probably changing conditions in himself as well as in his environment.

In the present research it became clear on these grounds that the hearing of all children suspected of aphasia should be tested. The next step then was to evolve a new form of test which would be effective where the 'routine' tests had broken down.

PART ONE

INVESTIGATION OF HEARING CAPACITY

CHAPTER II

HEARING TESTS, VALUE OF THE AUDIOMETER

PSYCHOLOGICAL DIFFICULTIES

WHY were the 'routine' tests ineffective with these children? In the course of the investigation two groups of reasons emerged:

(*a*) psychological, and

(*b*) of a physical nature.

The psychological reasons were the more obvious. Apart from the examination of the tympanic membranes and shadow cast by the ossicles, with lamp, mirror, and speculum, and the condition of the nose and throat, which are purely objective tests, the current methods of testing hearing involve understanding of instructions, a high degree of mental concentration, and very careful introspective judgement on the part of the patient. In tests with a tuning-fork the patient is required to understand that he is to listen to its sound, decide exactly when the sound has ceased to be perceptible to him, and indicate that moment by some signal, verbal or otherwise.

It was not a practical proposition to give these tests to children in the condition described in Chapter I. Frequently they failed to respond in any way to very loud sounds, and it was most unlikely that the soft pure tones of a tuning-fork would arouse their interest. Their 'bird-wittedness' made it extremely improbable that they could be induced to attend closely and for a sufficient length of time to decide at what moment the least perceptible intensity of a pure tone had been reached. The difficulty of giving the instructions was in fact insurmountable.

Actually it is usual in such cases to watch for dilatation of the pupils in response to the sounds used as test stimuli. But since any strong stimulus or an emotional condition of anxiety brings about the same reaction it was not to be relied upon, especially with children of such strongly emotional tendencies.

The subsidiary tests of the response to the tick of the watch,

Galton whistle, or acoumeter were open to the same objection, least of all perhaps in the case of the Galton whistle, which after-events have shown as potentially effective, though inferior to the audiometer. The human voice and whispered speech evoked no response (see pp. 1 and 63).

The physical reasons for the failure and inadequacy of the routine tests were found to be equally numerous.

It is necessary in this connexion to summarize very briefly the various tests of hearing which have been used in medical practice and for scientific purposes. They fall naturally into two groups:

(i) Those whose object is localization of the site of disease or injury of the auditory apparatus by direct methods.

(ii) Tests of auditory acuity.

Of the tests under (i)—apart from the examination of the appearance of the ears, nose, and throat, as already described —those named after Rinne and Schwabach have for a long period been treated as the most important and are still in current use.

THE RELATIVE AIR AND BONE CONDUCTION TESTS

'Rinne's Test (Combined Testing of Air and Bone Conduction).—If one holds a vibrating tuning-fork against the mastoid process or other portion of the skull until the tone is no longer heard (because the sound dies away), and then brings its prongs near the auditory orifice, the tone will again be perceived if the hearing is normal. The normal ear, therefore, hears the tone of the fork longer through the air than through the cranial bones. This is called the positive Rinne test'. Rinne stated that in those disturbances of hearing in which the tone of the tuning-fork is perceived stronger and longer through the cranial bones than when held in front of the ear, a disturbance in the sound-conducting apparatus exists. This is called the negative Rinne. 'Where, on the other hand, the tone of the tuning-fork is perceived longer before the ear than through the cranial bones (positive Rinne), we may conclude, according to Rinne, that the sound-perceiving apparatus is diseased.'[1]

'Schwabach was the first to find, in obstructions in the

[1] Politzer, 157.

sound-conducting apparatus due to diseases of the external or middle ear, that a vibrating tuning-fork, brought into contact with the cranial bones, is heard longer than under normal conditions, but that, on the other hand, the perception is shortened in diseases of the auditory nerve.'[1] The method adopted for giving this test is for the physician to find whether he or the patient can hear the tone of the fork, as the loudness decreases, for a greater length of time.

CRITICISMS OF THE RELATIVE AIR AND BONE CONDUCTION TESTS

Somerville Hastings, in his Presidential Address to the Section of Otology, Royal Society of Medicine, London, in November 1928 said: 'Our comparative ignorance concerning both the pathology and treatment of so many cases of deafness of both the middle and internal ear types is due, at any rate in part, to the methods of examination which we have used in the past.'[2]

Clarke, of the Ferens Institute of Oto-laryngology, in his paper 'Hearing Tests' at the same meeting, stated that 'These Tests (Rinne's, Schwabach's, and those based on similar principles) form the backbone of our functional diagnosis, and I believe that the lack of otological progress in the last thirty years is due to their inherent inaccuracy'.[3] He pointed out that some authors suggested that there were eight possible results of Rinne's test, that the test was originally based on the fallacy that for tuning-forks 'normally perception by air conduction is twice as long as by bone conduction' and that though this position was modified later by Rinne himself 'the point of change from a positive to a negative Rinne is only an arbitrary point on a sliding scale'.[4]

THE 'ABSOLUTE BONE CONDUCTION' TEST

'Since auditory ability may be considered as being the total hearing which is the result of the co-operation of conductive (external and middle ear) and perceptive (meaning here internal ear, nerve, and brain centres) components, it is obvious that by determining the condition of any two of these we can

[1] Politzer, 153. [2] Hastings, 73. [3] Clarke, 85. [4] Ibid. 86.

deduce the condition of the third. The Rinne and associated tests fail in their purpose of locating an auditory defect because their results are influenced simultaneously by changes in both components of the auditory function, conductive and perceptive.'[1]

The method now used by Somerville Hastings, Clarke, and others[2] is based on 'the absolute bone conduction test'. 'The observer (who presumably has normal hearing) and the patient each blocks his external auditory meatus with his finger (preferably by pressure on the tragus). A vibrating tuning-fork is then alternately pressed on to the mastoids of the patient and observer until it is no longer heard. In this way the bone conduction of the two persons is compared.'[3]

The strongest evidence adduced to show that the influence of the external and internal ear is eliminated by this test is that 'almost complete destruction of the drum, disruption of the ossicular chain or the occupation of the tympanum by fluid or organized contents causes no appreciable difference to perception by bone conduction so tested'.[4]

Somerville Hastings says: 'I have never yet found *absolute bone conduction* increased above the normal in any single case.'[5]

Having determined the condition of the patient's perceptive hearing capacity in this way, the condition of the conductive component can be reached if a comparison between the patient's total hearing (i.e. by air conduction) and the standard of normal total hearing is also made.[6]

QUANTITATIVE TESTS OF AUDITORY ACUITY

Somerville Hastings, in the Presidential Address already quoted, said: 'What I want to know before I can form a correct mental picture of a given case of deafness is, for every pure note between the upper and lower limits of audition, the percentage of normal hearing that remains. Then if we can also estimate in percentages the degree to which the cochlear nerves and cerebral centres are functioning, we shall be able

[1] Clarke, 89. [2] Ibid. 90. [3] Hastings, 75.
[4] Clarke, 90. [5] Hastings, 75. [6] Ibid. 77.

to say to what extent the deafness is due to changes in the conducting apparatus, and to what extent in the perceptive.'[1]

The aim of the otologist, therefore, is the measurement of a patient's auditory acuity, whether by air or by bone conduction, in terms of normal hearing.

The first essentials are a means of producing sounds whose frequency and intensity can be easily and accurately varied and controlled, and a standard or standards of normal hearing, based on actual tests of large and representative groups of individuals by these means.

In the present research the greatest need was to be able to test hearing for speech. It will be shown later that the range of the sounds produced in speech is wide, as regards both frequency and intensity. It has been thoroughly and minutely explored. As might be expected on biological grounds, hearing within this range is most sensitive and least susceptible to individual variations apart from the effects of disease or injury (see p. 51).

The apparatus, the methods of testing individuals and of obtaining a normal standard of hearing based on objective and quantitative data, employed in this investigation, are discussed below.

From the results came the discovery of a type of deafness which appears to have been hitherto unknown to otologists, and certainty of diagnosis among patients whose hearing capacity had been so difficult to estimate by the usual methods that it was in the first place unknown whether they had normal hearing or had been totally deaf from birth.

In the past, tests of auditory acuity, i.e. of total hearing capacity irrespective of the condition of the ears, have been made with three types of sound,

(*a*) noises, such as the ticking of a watch,

(*b*) the human voice or whisper,

(*c*) pure tones.

Early workers separated noises from tones and considered that they were perceived in a part of the labyrinth outside the cochlea.[2] It is now proved that noises are complex groups of notes of different frequency and intensity.[3]

[1] Hastings, 74. [2] Bunch 48. [3] Miller, 22.

(*a*) NOISES

Experimenters with noises as a source of sound for hearing tests have always found difficulty in controlling both intensity and quality. The watch has been and is widely used. It was recommended in the report of the Committee of Otological Research of the Royal Society of Medicine, 1918, also Politzer's acoumeter, a device for producing a standard noise of a 'ticking' nature.

For the purposes of the present research these were unsuitable. So far back as 1871 Wolf discovered that hearing acuity for the sound of a watch was not always correlated with hearing acuity for speech.[1] We know now that this is probably a matter of individual differences in acuity over different regions of the tonal range (pp. 27, 37 and 51). Somerville Hastings says that the watch and Politzer acoumeter 'though of value clinically are quite useless for scientific purposes'.[2]

Other attempts to standardize noises have been made with a pendulum and a falling weight.[3] I considered the potential value of the latter device, but it was clear that sounds of known relationship to those of speech must be sought if they could be found.

(*b*) THE HUMAN VOICE AND WHISPERED SPEECH

'There is no doubt that human speech, could it be definitely controlled, would furnish the most adequate and most comprehensive means of determining auditory acuity', wrote Bentley.[4] The importance of such a test if it could be fully controlled and standardized is clear. It would measure the response of the individual as a whole to the most important form of sound stimulus—the spoken word.

The capacity to hear speech has been shown in Chapter I to be essential to development of the speech-language function —the human characteristic.

The patients whom I was investigating (Chap. I) appeared deaf to speech before they received special teaching, but as they responded to teaching they began to recognize words by ear alone (pp. 63–6).

[1] Politzer, 148. [2] Ibid. 74. [3] Bunch, 51. [4] Ibid. 45.

I conceived the possibility of an age-scale similar to the Binet scale which would indicate the degree of development of any child with regard to speech heard.

The report of the Royal Society of Medicine 1918, already quoted, stated that 'A scheme for the tests of hearing was discussed and drawn up as follows: Conversational voice, single words or sentences with the opposite ear closed. Distance from each ear expressed as a fraction with the distance at which voice is heard from the normal ear as denominator. Whisper—conditions as above, but whisper produced by residual air after forced expiration. It is recognized that the voice tests are approximate only, as it is impossible to devise an exact standard. The words or phrases to be recorded when possible.'[1]

In this research, however, an 'exact standard' was needed.

(c) THE PHONOGRAPH AUDIOMETER

An early attempt to standardize tests of hearing for speech by means of the phonograph was made by Bentley, but was apparently unsuccessful.[2]

In 1927 Bell Telephone Laboratories (U.S.A.), in co-operation with the American Federation of Organizations for the Hard of Hearing tested 250,000 school-children by means of the phonograph audiometer. 'In this instrument records which have been made by special electrical processes are used for reproducing the speech sounds. This ensures a definite volume of voice, so that accurate comparisons are possible. The recorded speech sounds are transformed into their electrical equivalents by means of an electro-magnetic reproducer. The electrical waves thus created are carried by means of wires to a receiver which is held to the patient's ear. The intensity of the sound sent into the patient's ear is controlled by electrical resistances . . . with a single phonograph unit . . . and 40 receivers the pupils could be tested at the rate of about 100 an hour.'[3]

This apparatus, however, would not have been suitable for my own purpose. If the methods of testing evolved were to be effective for the most important purpose of all—diagnosis of young children in an 'aphasic' or deaf condition at the

[1] Bunch, 45. [2] Ibid., 48. [3] Fletcher, H., 211–13.

earliest possible age—the test stimuli must be designed to meet their total or almost total lack of ability to hear and understand words. I observed that normal young children sometimes found it difficult to interpret the sounds of speech even on the best gramophone. Without actual experiment it was impossible to say whether this was due to mechanical distortion of the actual sounds or to their originating in a machine instead of a visible responsive human personality.

(*d*) THE TONOSCOPE AND SENSITIVE FLAMES

From such mechanical reproductions of speech sounds I turned to the possibility of using the actual voice and speech of several individuals, who might be trained to repeat the same words or sentences with the least possible variation in speed, pitch, intensity, and pronunciation. For instance, tenor, baritone, soprano, and contralto voices might be represented. The speech used could be recorded during each test by mechanical means. The Westinghouse Co. had produced a 'tonoscope'—the sounds of speech spoken into a telephone receiver are transmitted to an electro-magnetic diaphragm carrying a minute mirror which by deflecting a beam of light traces a path either on a mirror or photographic film.[1] Results of later experiments made the use of this apparatus unnecessary.

Since the total intensity of the speech sounds used seemed an important factor in determining the response of the patients to speech heard—after they had begun to progress with special teaching—I spent a great deal of time in an attempt to find a type of sensitive flame apparatus which would give a constant reaction to the total intensity of sounds produced at any point of the maximum tonal range involved in speech.

This attempt was to a large extent successful. Using an 'upright' gas burner with the 'top' taken off as described by Barton,[2] with water manometer to measure the gas pressure, a choke with indicator on the leading-in pipe, and another indicator on the adjustable control of the burner itself, and with other details also standardized, I was able to produce a

[1] Described in a pamphlet issued by the Company.

[2] Barton, 365.

flame which reacted to the pure tones produced by the audiometer (p. 24 below) with a degree of regularity.

This apparatus was afterwards discarded. Even in the laboratory slight sounds from outside, if of high frequency, were apt to disturb the flame. Further, the audiometer tests evolved later, corroborated by other tests, proved that recourse to human speech or a whisper, with their inevitable train of individual differences affecting both the sounds uttered and the reaction of the listener, is unnecessary.

(*e*) PURE TONES

Early workers, such as Scheibler (1834), Koenig, and Bezold,[1] realized the need for testing auditory acuity at representative intervals of frequency over the entire range of audibility. Their chief form of apparatus was the tuning-fork. The monochord, whistles, and musical instruments have been and are used to produce sounds of frequency and intensity for which the tuning-fork is unsuitable.

DIFFICULTIES IN THE USE OF TUNING-FORKS

There are four main sources of difficulty in the use of the tuning-fork as an exact quantitative test of auditory acuity.

First, the intensity of the sound produced is extremely difficult to control. Various workers have sought mechanical means of giving a 'standard blow', by using a falling weight (Downey),[2] or a trigger attachment (Gradenigo).[3] Devices have also been used for observing the excursions of the prongs through a narrow slit in a plate of attached metal, and for graphically recording the decrement of vibration of the fork.

These methods have proved so little satisfactory that a completely different principle is now adopted—the calculation of 'hearing distance'. When the patient has ceased to hear the sound of a vibrating fork, a stop-watch is used to record the number of seconds for which the sound can still be heard (as it dies away) by a normal ear. On the principle that the intensity of a sound varies inversely to the square of its distance the patient's percentage retention of normal hearing distance is calculated.[4] The damping constant (rate of decrement) of the fork should be known.

[1] Bunch, 49. [2] Ibid. 49. [3] Ibid. 50. [4] Clarke, 97–102.

The calculation of hearing distance, therefore, involves a second difficulty, the establishment of the standard of normal hearing. At the present time no such standard of normal hearing, based on the use of a definite range of particular forks, can be said to exist. The individual worker is compelled to establish his own standard of normality, and he may have to use his own or an assistant's auditory acuity as the actual basis of comparison.[1]

Unless there is gross deafness, therefore, tests of auditory acuity, in fact all tests with the tuning-fork or similar apparatus, involve a strong personal factor. Actual tests of auditory acuity by purely objective methods show, that among individuals whose hearing is not known to have been affected by either disease or injury, differences in auditory acuity all over the tonal range are often considerable (see pp. 27, 36 and 51). The hearing of no two individuals is the same.

The third difficulty in the use of tuning-forks is their limited range of intensity. In this research it was found that children incapable of hearing the loudest sounds producible by the tuning-fork, or indeed any of the apparatus used hitherto, had hearing, whose value, though limited, was afterwards definitely established (p. 73).

It is clear that the maximum range of intensity in tuning-forks by no means covers the complete range of normal hearing for intensity (p. 26).

Fourthly, methods of testing hearing by means of tuning-forks, if they are to indicate the individual's hearing capacity for speech, are extremely cumbersome and protracted. The patient should be tested with at least 8 forks (see pp. 55–7), and rapid or relatively rapid changes in the intensity of the sound produced by each are practically impossible.

THE AUDIOMETER AS A SOURCE OF PURE TONES

Realizing that these difficulties prevented full, exact, and standardized tests of hearing by means of tuning-forks, a succession of workers have evolved a form of electrical apparatus known as the audiometer.

The first audiometer was apparently produced by Urbantschitsch in 1890.[2] A later and more important type was

[1] Clarke, 97. [2] Ibid. 95.

originated by Seashore in 1897. An improved model of this, called the *Iowa Pitch Range Audiometer*, the product of 'the efforts and co-operation of several departments (in the State University of Iowa), principally Otology, Physics, and Electrical Engineering with that of Psychology', was used for important investigations, the results of which were published in 1922.[1] The principal features are the absolute control of both pitch and intensity over a wide range. Two generators, one for low and one for high frequencies are operated by an electric motor and produce pure tones in a carefully standardized telephone receiver which the patient or observer holds to one ear at a time. Variations in intensity are obtained by resistance coils. There are 38 variations in pitch, from 30 v.d. to 14,500 v.d., and 22 steps in intensity. The latter are purely arbitrary and based on a scale of resistance to give increments of 250 per cent.

More recently the Western Electric Company, U.S.A., has produced a series of audiometers on the basis of fifteen years of research by Bell Telephone Laboratories.[2] This research involved the most thorough and scientific investigation of hearing capacity which has yet been made by any workers.

INVESTIGATION OF HEARING BY BELL TELEPHONE LABORATORIES

The leader of the group of workers associated with Bell Telephone Laboratories is Harvey Fletcher. In reaching his final conclusions he made use of additional data, obtained on methods similar to his own, by independent investigators, Wien, Kranz, and Lane.

In this series of experiments the hearing capacity of about 100 men and women, any with defects in hearing having been eliminated,[3] was tested in a most exact manner.

(*a*) STANDARDS OF INTENSITY AND OF NORMAL HEARING

The threshold of audibility or minimum perceptible intensity for pure tones, over the entire range of audible frequencies, was found by means of apparatus involving 'vacuum tubes (in the form of oscillators, amplifiers, and rectifiers),

[1] Bunch, 59–67, and Zuehl, 83. [2] Fletcher, H., l. c.
[3] Ibid. 137.

condenser transmitters, and attenuators accurately calibrated throughout a wide range of intensity'.[1] The ultimate means of calibration was the thermophone, a device which makes it possible to calculate sound energy in terms of voltage. The problem which workers with tuning-forks have sought to meet by means of a standard blow has thus at last been solved. Moreover, the normal threshold of audibility was calculated by Fletcher from actual tests of a large group of men and women, on this uniform basis and under identical conditions, thus meeting the second difficulty in the use of tuning-forks.

(*b*) THE 'THRESHOLD OF FEELING'

A definite upper limit of audibility for intensity was discovered by these workers. 'When a tone exciting the ear is continuously increased in intensity, it finally reaches a loudness which produces the sensation of feeling. If the tone gets much louder than this value, it becomes painful. Although this threshold of feeling may have no relation to the auditory sense, it does serve as a practical upper limit for the intensities of tones which can be sensed by the ear.'[2] This sensation is described as at first a tickling sensation and then as actually painful in the middle ranges of hearing. In the lower frequency range it is a flutter. At the upper limit of audible frequencies 'it is very difficult to distinguish between the sensation of feeling and the sensation of hearing'.[3]

(*c*) UPPER AND LOWER LIMITS OF AUDIBLE FREQUENCY

The average values of the upper and lower limits of audible frequency were found to be 20,000 v.d. as the upper limit and 20 v.d. as the lower.[4]

(*d*) THE 'AUDITORY SENSATION AREA'

Using octave intervals of frequency (8 to 16,384 v.d.) as his abscissae, and a logarithmic scale of intensities based ultimately on electrical voltage as his ordinates, Fletcher was able to produce a graphic record of the whole average 'audi-

[1] Fletcher, H., 134. [2] Ibid. 142. [3] Ibid. 143.
[4] Ibid. 144.

tory sensation area'. The average thresholds of hearing and feeling appear as smooth curves furthest apart at the frequency 1,000 v.d. and meeting at the average upper and lower limits of audible frequency.[1]

The minimum perceptible differences in frequency and intensity were also investigated. In intensity the ratio of the smallest perceptible difference in any part of the auditory sensation area to the largest was 0·21 to 9.[2] In pitch the number of perceptible gradations found was approximately 2,000.[3]

THE NO. 2A WESTERN ELECTRIC AUDIOMETER

The audiometer used in the research described in this book is a Western Electric Co.'s No. 2A Model, the property of the Department for Training Teachers of the Deaf at Manchester University. The ultimate source of sound is a special telephone receiver, which the patient holds to his ear. Any risk of inaccuracy in calculating 'hearing distance' (p. 21) is thus eliminated and 'is not subject to variations in the acoustic properties of the room'.[4]

The generator is 'a special vacuum tube oscillator designed specially for producing pure tones. . . . Eight octave tones spaced an octave apart in the frequency range, from 64 to 8,192 cycles (double variations) per second, can be produced by throwing the appropriate keys on the top of the audiometer'.[5] It will be shown in Chapter IV that this represents the range of frequency involved in the sounds of human speech. There are 25 steps in intensity. The average normal threshold of audibility calculated by Fletcher after the experiments already described (p. 25) is marked 0. 'Hearing loss' is indicated if the intensity of any tone has to be raised to any of the steps marked 5–100 in order to be just perceptible to the patient. Individuals with supernormal sensitivity to sound are able to hear tones when the intensity is reduced to the steps −5 to −20.

It is important to remember, when using this audiometer that Fletcher's standard of normal hearing was calculated from tests of adults in 'sound-proof booths'.[6] 'Room-noise'

[1] Fletcher, H., 157. [2] Ibid. 159. [3] Ibid. 162.
[4] Ibid. 214. [5] Ibid. 216. [6] Ibid. 137.

is present in any ordinary building and may greatly affect results of hearing tests.[1] Calculations from actual experiments show that the average 'hearing-distance' for speech in a 'sound-proof booth' and a 'quiet office' are 1,250 ft. and 125 ft. respectively.[2]

The loudest sound producible by the No. 2A Audiometer is considerably below the 'threshold of feeling'. The range of intensity covered is equal to 70 per cent. of the average normal auditory sensation area calculated by Fletcher.[3] If the patient is just able to hear a tone, therefore, at the step of 'hearing loss' marked 100 the actual percentage of hearing that remains, between the upper and lower limits of audition, for that tone, is 30 per cent. The Western Electric No. 1A audiometer, a much larger instrument, covers approximately the whole normal auditory sensation area.[4]

The steps in intensity in the No. 2A audiometer are based on calculations of 'sensation units'.[5] These have been criticized as arbitrary. They are actually logarithmic units of sound energy translated into terms of voltage by the thermophone.[6] They form a most convenient scale of intensities between the lower limit of audibility and the upper threshold of feeling, exactly as the number of degrees between freezing- and boiling-point are measured on the thermometer. They were calculated, moreover, after actual measurements of the average intensity of human speech as well as of hearing capacity.[7]

Changes in intensity in the No. 2A audiometer are produced by the simple turn of a dial. The instrument as a whole is most easy to use and readily portable.

Used in the light of the conclusions reached by Fletcher and the other workers mentioned, it was clear that an audiometer was the most accurate and scientific apparatus available for making tests of hearing. The difficulties of tuning-forks were obviated (p. 23).

Though the No. 2A Western Electric Model has no facilities for testing deafness greater than 70 per cent. this disadvan-

[1] Fletcher, H., 101. [2] Ibid. 187.
[3] See pamphlet issued by Western Electric Co., also Fletcher, H., 216. [4] Fletcher, H., 157, 201, and 216.
[5] Ibid. [6] Ibid. 134. [7] Ibid. 68.

tage, as compared with the No. 1A and audiometers of other types producing pure tones, is partly outweighed by its portability. The extreme lower and upper limits of frequency are not covered by it, but they are not involved in human speech sounds (pp. 26 and 56), and to test them proved unnecessary for diagnosis during this research.

Actually the form of test adopted proved successful beyond expectation. It showed that the apparently variable response to sound of two of the children described in Chapter I was due to an unusual and unsuspected condition of the auditory function. The difficulty of diagnosis by other methods was explained.

But though this apparatus would give quantitative measurements of the hearing of the patients in terms of frequency and intensity, the psychological difficulties in part due to their particular condition and in part to their age had yet to be overcome.

THE TUNNEL APPARATUS

It was necessary to induce these young children to listen with the closest attention to pure musical tones which experience had shown not naturally interesting to them.

Before special teaching they had rarely, as compared with the normal child, responded to sound: since its inception they had begun to acquire such habits, but these were recent and at first functioned in a limited number of environmental conditions. I had to provide them with a cause for the sounds used, consistent with their restricted previous experience. I needed to give them practice, under controlled conditions, to counterbalance their lack of auditory experience and to ensure that they were reacting in the required manner to the correct stimulus. It was necessary to provide them with an interesting method of response.

Accordingly, what I have described as the 'Tunnel Test' was evolved. There are also complementary tests described in Chapter IV.

The apparatus for the Tunnel Test consists of a section of model railway 4 ft. long, the whole modelled and coloured like a tunnel and built up in a portable wooden case. This is placed on the table at a distance of about 2–3 ft. from the

child. In the middle of the surface facing the child is an opening 8 in. by 6 in., through which the railway track is visible. There is also a smaller aperture in the middle of the reverse surface of the tunnel. Showing through this aperture is a 150 watt gas-filled lamp which is otherwise completely enclosed. The lamp, therefore, when lighted illuminates the tunnel, which is so placed as to be in shadow.

The lamp is operated by a key placed in front of the child. There is also a model electric locomotive placed inside the tunnel, and at the beginning of the test invisible to the child. The experimenter has, in the lid of the audiometer, a tumbler switch marked L (lamp only) and E (engine). When the switch is in the E position, the result of depressing the key is, that the lamp is illuminated and the engine begins to move and then passes the opening in front of the tunnel, so being visible to the child. Half-way along the length of track the current is cut off from the live rail by a piece of vulcanite insulation. The engine, therefore, loses speed and is stopped by a block of wood at the end without being derailed. It can then be drawn back to the starting-point with a piece of string by an assistant. If, however, the switch is in the L position when the key is depressed the lamp is illuminated only.

This apparatus can be worked from the ordinary electric light supply (200 or 240 volt D.C. mains), using the lamp as a transformer as well as a signal. We have also arranged it to work from 6-volt accumulators with a low resistance engine and lamp.

The methods and procedure of the Tunnel Test, using this apparatus[1] in conjunction with the audiometer, are discussed in the next chapter.

[1] I am indebted to F. S. Wilson of the Physiology Department, Manchester University, for his assistance in planning the rather complicated wiring of this apparatus and for constructing it from my directions.

CHAPTER III

HEARING CAPACITY OF NORMAL, APHASIC, AND DEAF CHILDREN

SINCE the children for whom the test was particularly intended would be either without speech and language or only in process of acquiring it as a result of special teaching, it was desirable to make their part in the test clear to them from the nature of the visible situation and to be able to give the minimum of explanation by means of gesture if necessary, though in testing children who could understand speech it would be unnatural not to use words. The child's comprehension of the situation and the instructions must be tested in the practice before the test proper.

THE TUNNEL TEST

Object.

Exploration of minimal intensity threshold for frequencies 64, 128, 256, 512, 1024, 2048, 4096 and 8192 v.d., with children.

Need of an Assistant.

With older children it is sometimes possible for an experimenter to carry out this test without the help of an assistant. As a rule, however, two trained workers are required. One controls the audiometer and L/E switch (p. 30) and records results. The other shows the child what to do and observes his reactions: a task requiring considerable training and experience with all, and especially unusual, types of children. The apparatus here described has been found to reduce the possibility of 'wrong answers' very considerably, but the manner and method used in demonstrating the test to the child will largely determine the amount of interest, care, and confidence with which he performs it. With children unaccustomed to hearing or deficient in language the observer must be on the watch for incorrect responses by the child, although the safeguards provided by the apparatus and procedure, detailed below, have proved a very effective means of checking inaccuracies.

ARRANGEMENT OF APPARATUS

Child seated at table, on which is placed tunnel, in front of him and 2–3 ft. distant. Key is immediately in front of him. Audiometer, L/E switch, and note-book are behind his back.

PREPARATORY PRACTICE AND INSTRUCTION

Experimenter sits down beside child. Points out rails inside tunnel. Shows child that tunnel can be illuminated by depressing key (the L/E switch has been previously set in L position so that locomotive remains stationary). Child is taught to work key correctly first with right then with left hand. Experimenter takes up receiver and holds it to his ear, suggesting, especially by facial expression and attitude, that he is listening; he points at the same time to the railway lines. If child is capable of understanding he is asked, 'Have you wireless at home? This is like wireless. You can hear the train coming.' At first experimenter indicates that he can hear nothing. He has his right forefinger on the key. Assistant, behind back of both child and experimenter, switches on a note of audiometer. When experimenter hears this note he indicates the fact to the child by facial expression and/or speech. He depresses the key, illuminating the tunnel. The assistant waits 1–3 seconds, then moves the L/E switch to the E position thus releasing the locomotive which traverses the tunnel and is seen by the child through the aperture (p. 30). The purpose of the delay, which should vary in length of time, is to prevent the child suspecting that he can set the locomotive in motion by depressing the key. He is to understand that he must illuminate the tunnel when he hears the sound in the telephone receiver. While the L/E switch is in the L position the locomotive will not move, and during the instruction and preliminary practice the assistant starts it by releasing this switch. During the actual test the switch remains in the E position throughout, so that the child himself starts the locomotive each time he depresses the key and lights the lamp. The advantages of this arrangement are that no sound caused by the movement of the engine can interfere with the effect of the test stimulus, and that there is no loss of time (pp. 28, 35).

The demonstration by the experimenter should be given

twice. The child is now shown how to hold the receiver in his right hand with the exposed part of the diaphragm opposite the external auditory meatus. The demonstrator says or pantomimes, 'Watch' and 'Listen, the train is coming', the note 1024 v.d. is sounded, loudly, say at 70 on the 'loss of hearing' dial (see p. 27), or at the maximum if the child has been judged to have very little hearing, in order that there may be no question in the child's mind about the existence of the sound. Often there is a sudden and marked change in the child's facial expression, directly he hears a loud sound, and a quite definite change for a softer sound in this test. If the child shows this reaction at the first practice, but omits or hesitates to press the key, he is reminded to do so by the experimenter.

In subsequent practice-sounds the note 256 v.d. is also used to accustom the child to responding in the same way to whatever note he hears in the receiver, irrespective of frequency.

If the child proves unable to hear 1024 v.d. at the maximum intensity other frequencies are successively used until, if possible, a reaction is obtained. The object at this stage is to make certain that the child can hear 1024 v.d. or, failing that, other frequencies, and to train him to press the key immediately he can hear any sound in the receiver.

The next step is to obtain responses to notes of minimal intensity. The 'loss of hearing' dial is now set at '0'. In the few instances during this research in which children have heard any frequency at this intensity the note has been promptly repeated using the 'supernormal' step '20' (p. 27) as the starting-point.

The principle employed is to proceed from 'sound not heard' to 'sound heard'. Results have proved that this is much more effective than the reverse principle with children. The intensity (i.e. 'loss of hearing') dial is now revolved to give increases in intensity at the rate of one step (e.g. from '5' to '10') per second. If the earlier practice has been efficient the child will react by pressing the key directly he can just hear the sound. The number of this step (e.g. 5, 25, 50, &c.) represents the child's minimal threshold for that particular frequency. Occasionally, with abnormal or very deaf children unreliable results at this stage indicate the need

for more training by the earlier methods. Usually, however, two repetitions of each note, 1,024 and 256 v.d., with each ear have been sufficient to train the child in the procedure.

The practice series therefore are

(1) Right ear, 1,024, 256.
(2) Left ear, ,, ,,

TEST

Series (3) Right, 128, 512, 2,048, 4,096.
,, (4) Left ,, ,, ,, ,,
,, (5) Right, 64, 256, 1,024, 8,192.
,, (6) Left ,, ,, ,, ,,

(This arrangement was originally prepared so that Series (5) and (6) might be omitted and yet sufficient data be obtained to evaluate an observation described in Chapter IV, p. 58.)

After the practice the tumbler switch may be left in E position.

Each frequency is given precisely as in the latter part of the practice, intensity being raised from 0 by single step increases at 1 second intervals in each case until the child responds.

CHECKS OF RESULTS

(1) *Improbable but possible results.*

When the child has responded to widely different intensities for neighbouring octaves, e.g. at step '25' for frequency 128 v.d. to step '60' for 256 v.d. and to step '25' for 512 v.d., he is given at least two repeat tests to test the doubtful reading or readings. As the data provided below indicate, the readings when presented in graph form usually show a simple or a complex curve.

(2) *Mistakes.*

Sometimes reaction takes place when no sound is being produced; this may be due to over-expectation or to unavoidable clicks occasionally occurring at changes of contact during movements of the intensity dial. A child suspected of responding without hearing sounds may sometimes be detected by the experimenter's refraining from switching on a note but appearing otherwise to continue the test. An apparently careless child is sometimes asked (if conversation

is possible) to describe the sound in terms of his experience. A nervous or uncertain child may be given sounds of unmistakable intensity to build up his confidence.

(3) *Repetitions.*

These may be required to check improbable results as described above.

In estimating the correctness of the results, if two intensity readings for the same frequency show a difference of two or more steps, a further reading is taken. In cases of two intensity readings with a smaller difference, the response to the lower reading has been adopted. With three varying readings, the two alike or the middle one of three (not more than three steps apart) have been accepted. Sometimes more than three readings of the same frequency are necessary. But as a rule repetition confirms the first reading within a limit of one step either way, even when the whole test is repeated on different dates.

(4) *Final Check.*

To be given when child is not too fatigued, or on another day. The whole test is repeated, but each frequency is sounded only at the intensity to which the child previously responded. In cases of no response to any sound used during this check, the intensity is increased until the patient reacts.

IMPORTANT NOTE.

It is essential whenever possible to prevent the child's discovering that he has not heard a particular note. Therefore, when he cannot hear a given frequency at the maximum intensity, the experimenter immediately uses another.

In certain forms of deafness response to a given intensity of a note has been found to be obtainable after a delay of even 5 or 6 seconds' duration. Such patients require a modified procedure.

Fatigue, especially as affecting attention, must be watched for, and if necessary rests given.

The whole test has been designed so that the time occupied shall not exceed 15 to 20 minutes. This appears to be the maximum period suitable for young children, and especially children whose hearing and/or language capacity are deficient.

LAMP SIGNAL

A lamp signal, lighted by pressing a button-switch, which the patient holds in the opposite hand to that used for the receiver, is provided with the No. 2A audiometer. With older boys and girls this may be substituted for the tunnel apparatus. Where it has been used in the following tests the fact is recorded.

CHANGES DUE TO PRACTICE, ETC.

The possibilities of changes due to practice and training, health and weather conditions, were investigated in a later series of experiments (pp. 70–4.)

HEARING CAPACITY OF NORMAL CHILDREN

In order to make a sample standardization of the 'Tunnel Test' permission was obtained from the Senior Medical Officer, Dr. Ritchie, of the Manchester Education Authority, and from the Head Mistress, to give the test to 12 children, aged 6–8 years, in the Mansfield Street Infant and Elementary Schools, Ardwick, on September 21st, 1928. The results below indicate the step of intensity at which the child reacted to each frequency.

The steps in intensity are marked:

—20, —15, —10, —5, 0, 5, 10, 15, 20, 25, 30, 35, 40, 45, 50, 55, 60, 65, 70, 75, 80, 85, 90, 95, 100, 105; —20 is the lowest intensity (softest sound) and 105 the greatest (loudest sound).

TABLE A. HEARING CAPACITY OF NORMAL CHILDREN

These results are presented in graphic form on p. 38.

Frequencies	64	128	256	512	1,024	2,048	4,096	8,192
Aged 6 yrs.								
No. 101. Boy	−15	−10	0	15	20	20	5	5
	0	−5	−5	−10	−10	5	5	0
No. 102. Girl	45	25	30	30	25	25	25	30
	30	30	30	40	25	15	20	20
No. 103. Boy	25	35	25	35	15	5	20	35
	−20	−20	−10	0	0	0	0	0
No. 104. Girl	20	20	25	25	0	5	5	10
	15	20	10	25	15	5	0	0

Frequencies	64	128	256	512	1,024	2,048	4,096	8,192
Aged 7 yrs.								
No. 111. Girl	35	30	40	40	35	20	25	10
	25	35	30	40	30	15	10	10
No. 112 „	25	20	25	30	30	10	10	5
	20	25	25	30	25	5	10	5
Aged 8 yrs.								
No. 105. Girl	25	25	25	30	20	5	5	20
	30	30	30	35	20	5	5	15
No. 106 „	30	25	25	40	40	35	45	30
	25	25	25	25	25	15	25	20
No. 107 „	20	15	25	20	25	0	10	10
	15	20	15	10	20	0	15	20
No. 108 „	50	30	55	30	35	20	10	30
	40	40	50	40	30	20	20	25
No. 109 „	20	20	30	30	25	10	5	20
	25	35	30	25	25	0	20	25
No. 110 „	30	25	25	30	25	10	20	15
	25	25	30	30	30	10	10	5

HEARING CAPACITY OF APHASIC CHILDREN

The principal discovery of this research was the existence of a hearing defect which I have termed 'high-frequency deafness' (see p. 41). Five 'aphasic' boys and one girl (Nos. 1–6) were found to suffer from this defect. The patient No. 7, who had been previously diagnosed as born totally deaf, was shown to have normal hearing. The proof of these conclusions is in the table of results given below, considered in relation to the analyses of speech sounds summarized in Chapter IV.

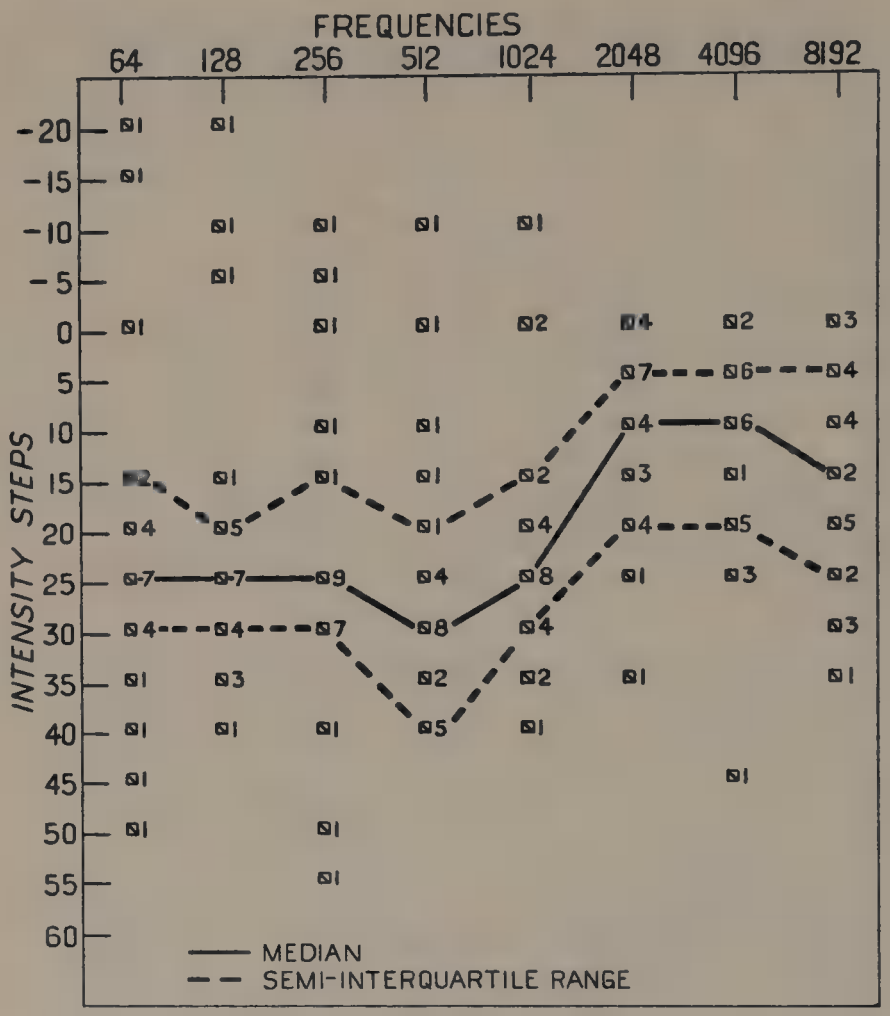
HISTORIES OF APHASIC CHILDREN

The histories of patients Nos. 1, 2, and 7 prior to this research were outlined in Chapter I.

I found four boys in the Royal Schools for the Deaf, Manchester, whose behaviour and previous histories were similar.

The Head Master of these schools, John Spalding, Esq., offered every possible facility for this investigation.

No. 3 was apparently deaf and could not speak on entering the school at the age of 9 years in April 1927. He had spent a fortnight under observation in a Psychological Laboratory,



Hearing Capacity of Normal Children tested with the Audiometer and Tunnel Apparatus. (Table A, p. 36.)

(Number of readings at each step is stated, e.g. 5.)

an establishment founded for the study of abnormal children, and was reported to have normal auditory acuity. His local Medical Officer, however, entered 'Born deaf' on his admittance sheet. There was 'bird-wittedness' in this case (p. 2).

No. 4 had a similar history, and at the age of 14 years was still abnormal in response to sound and in speech. 'Aphasia' was marked on his admission form.

No. 5 had entered the school in 1918, and was under Mrs. Ewing's observation for a period of 11 years. At first his behaviour approximated to that of Nos. 1, 2, &c. When I first began to observe him early in 1928 he could follow conversation in a rather loud voice (see test results on p. 64) by ear alone, and his speech was fluent and intelligible, though not perfectly normal.

Another patient, No. 6, born November 9th, 1913, was found during the testing of 71 deaf children described below. This boy in 1928 was described as 'peculiar' and there was great uncertainty as to the amount of hearing he possessed. I found conversation with him difficult, yet he responded better to noises than the usual 'partially deaf' child. 'Born deaf' was the description on his admission form.

At the same time I had also three other private patients exhibiting similar symptoms.

No. 8 first came under my care in October 1928 at the age of 7 years. His mother reported that he disliked conversation and as a rule avoided it. His speech before he came to me for special teaching was in the form of hurried and usually quite unintelligible jargon (see p. 135), resembling that used by No. 2 in 1926 (see p. 129). His brother, aged 4 years, spoke and conversed normally. This patient was reported to have begun to speak at the late period of 3–4 years old.

No. 9 has come to me for special teaching since October 1926. His progress was sufficiently good for occasional visits to be adequate since July 1927. At 4 years of age, his mother reported, his attempts to speak were quite unintelligible. When I first saw him at the age of 11 years it was almost impossible to understand what he said, and his language capacity proved later to be greatly below that of a normal boy of his age and social environment.

No. 10 was a pupil at a girls' boarding school of high

standing. She was sent to me at the age of 14 years for special teaching on account of inability to learn to read and write, although her education had been begun at the age of about 7 years. There were no obvious defects in hearing and understanding words, or in speech, but subsequent investigation (p. 119) showed that language development was retarded and that there was inability to repeat correctly passages of prose read aloud to her. I gave her courses of special teaching in 1926 and 1927 and tested her in January 1929. There was no history of serious illness or injury among the patients Nos. 8, 9, and 10.

The educability of 'aphasic' children was also investigated in this research, and the results which are possible, especially with the high-frequency deaf, are described in later pages (pp. 115–24 and 128–36).

TABLE B. HEARING CAPACITY OF APHASIC CHILDREN

(For order of readings, see p. 34.)

These results are presented in graphic form on p. 45.

	64	128	256	512	1,024	2,048	4,096	8,192
No. 1. Boy, 8 yrs.	20	35	35	60	65	65	nil.	nil.
	25	45	40	65	70	75	,,	,,
No. 2. Girl, 10 yrs.	35	35	35	55	90	70	,,	,,
	25	35	25	45	85	nil.	,,	,,
No. 7. Girl, 22 yrs. (see	15	15	10	20	5	5	10	15
pp. 2 and 121–4).	25	15	20	20	5	10	15	30
No. 3. Boy, 10 yrs.	45	35	50	65	70	65	75	nil.
	45	45	50	70	70	75	65	,,
No. 4. Boy, 14 yrs.	35	25	45	55	65	60	70	,,
	40	40	50	60	70	65	75	,,
No. 5. Boy, 14 yrs.	15	10	25	25	45	50	65	,,
	5	5	15	20	45	35	65	,,
No. 6. Boy, 15 yrs.	—	40	45	—	70	65	65	,,
	—	45	40	—	75	70	70	,,
No. 8. Boy, 7 yrs.	30	25	25	10	35	20	10	40
	40	20	35	25	25	15	15	40
No. 9. Boy, 15 yrs.	—	15	—	15	—	5	5	—
	—	10	—	15	—	0	15	—
No. 10. Girl, 17 yrs.	15	15	10	20	10	5	10	0
	15	10	15	15	20	0	5	5

Ages given at date of test.

Nil = no response to maximum intensity for this frequency.

— = not tested for this frequency.

COMPARATIVE HEARING OF NORMAL AND 'APHASIC' CHILDREN

The results in Table B indicated that there were two distinct types of hearing capacity among the ten aphasic patients. The readings of Nos. 7, 8, 9, and 10 for all frequencies fell well within the distribution for normal children (see Table A and pp. 66–7).

With the patients Nos. 1, 2, 3, 4, 5, and 6, however, there was an unexpected combination of features. The readings for their responses on the three lower frequencies, 64, 128, and 256 v.d., fell definitely within the distribution for children of normal hearing, but there was a severe defect of hearing for high frequencies. To this type of deafness, the existence of which appears to have been hitherto unknown, I have given the description 'high-frequency deafness'. The investigation of its effects on the development of speech and language is described in Chapter IV. The aetiology of the apparently aphasic condition of patients Nos. 1–6 was afterwards shown to be explicable in terms of this previously unknown form of defective hearing, while the condition of Nos. 7–10 was found due to another type of trouble.

The test, therefore, has provided a means of proving the two forms of defect distinct from each other. It enables a diagnosis to be made on a quantitative basis. But at the time, before adopting any conclusions, it was desirable to find out whether the condition of Nos. 1–6 was ever paralleled among children suffering from congenital or acquired deafness, but not presenting the symptoms of apparently variable response to sound, 'bird-wittedness', &c. Permission was therefore obtained to test a large number of deaf children in the Royal Schools for the Deaf, Manchester.

TABLE C. HEARING CAPACITY OF DEAF CHILDREN

Nil = no response at maximum intensity.

— = not tested for this frequency.

These results are presented in graphic form on p. 45.

	64	128	256	512	1,024	2,048	4,096	8,192
No. 201. Boy, 8 yrs.	50	55	55	60	75	80	80	nil.
	45	60	50	45	65	80	80	,,
No. 202. Boy, 8 yrs.	35	30	40	55	65	65	70	,,
(p. 65).	35	30	45	55	60	55	70	,,
No. 203. Boy, 8 yrs.	nil.	nil.	nil.	85	nil.	nil.	nil.	,,
	,,	,,	,,	nil.	,,	,,	,,	,,
No. 204. Boy, 8 yrs.	40	50	60	80	,,	,,	,,	,,
	45	40	55	nil.	,,	,,	,,	,,
No. 205. Girl, 8 yrs.	nil.	70	80	,,	,,	,,	,,	,,
	,,	nil.	nil.	,,	100	,,	,,	,,
No. 206. Girl, 8 yrs.	—	70	80	—	100	,,	,,	—
	—	70	85	—	105	,,	,,	—
No. 207. Boy, 8 yrs.	nil.	nil.	nil.	nil.	nil.	nil.	nil.	nil.
	,,	,,	,,	,,	,,	,,	,,	,,
*No. 208. Boy, 8 yrs.	—	5	—	10	—	5	0	—
	—	15	—	5	—	5	15	—
No. 209. Boy, 9 yrs.	nil.	nil.	nil.	100	95	90	nil.	nil.
	,,	,,	,,	100	100	90	80	,,
No. 210. Boy, 9 yrs.	,,	65	,,	100	nil.	nil.	nil.	,,
	,,	70	80	95	,,	,,	,,	,,
No. 211. Girl, 9 yrs.	—	nil.	85	—	95	,,	,,	—
	—	,,	80	—	90	90	,,	—
No. 212. Boy, 10 yrs.	nil.	,,	nil.	95	95	nil.	,,	nil.
	,,	,,	,,	95	105	85	,,	,,
No. 213. Boy, 10 yrs.	,,	,,	,,	90	nil.	90	75	,,
	55	40	50	40	35	20	20	10
No. 214. Girl, 10 yrs.	nil.	70	nil.	100	95	85	nil.	nil.
	,,	nil.	,,	100	90	80	,,	,,
No. 215. Girl, 10 yrs.	—	40	50	—	50	35	80	—
	—	60	60	—	70	65	70	—
No. 216. Girl, 10 yrs.	—	70	80	—	100	nil.	nil.	—
	—	70	85	—	105	,,	,,	—
No. 217. Girl, 10 yrs.	—	nil.	nil.	—	105	,,	,,	—
	—	,,	,,	—	100	,,	,,	—
No. 218. Girl, 10 yrs.	—	,,	,,	—	nil.	,,	,,	—
	—	,,	,,	—	,,	,,	,,	—
No. 219. Boy, 10 yrs.	nil.	,,	,,	nil.	nil.	,,	,,	nil.
	,,	,,	,,	,,	,,	,,	,,	,,
No. 220. Boy, 10 yrs.	50	50	55	55	60	85	80	,,
	nil.	65	80	95	100	nil.	nil.	,,
No. 221. Boy, 10 yrs.	45	40	55	55	60	55	75	,,
	55	35	80	95	105	70	nil.	,,

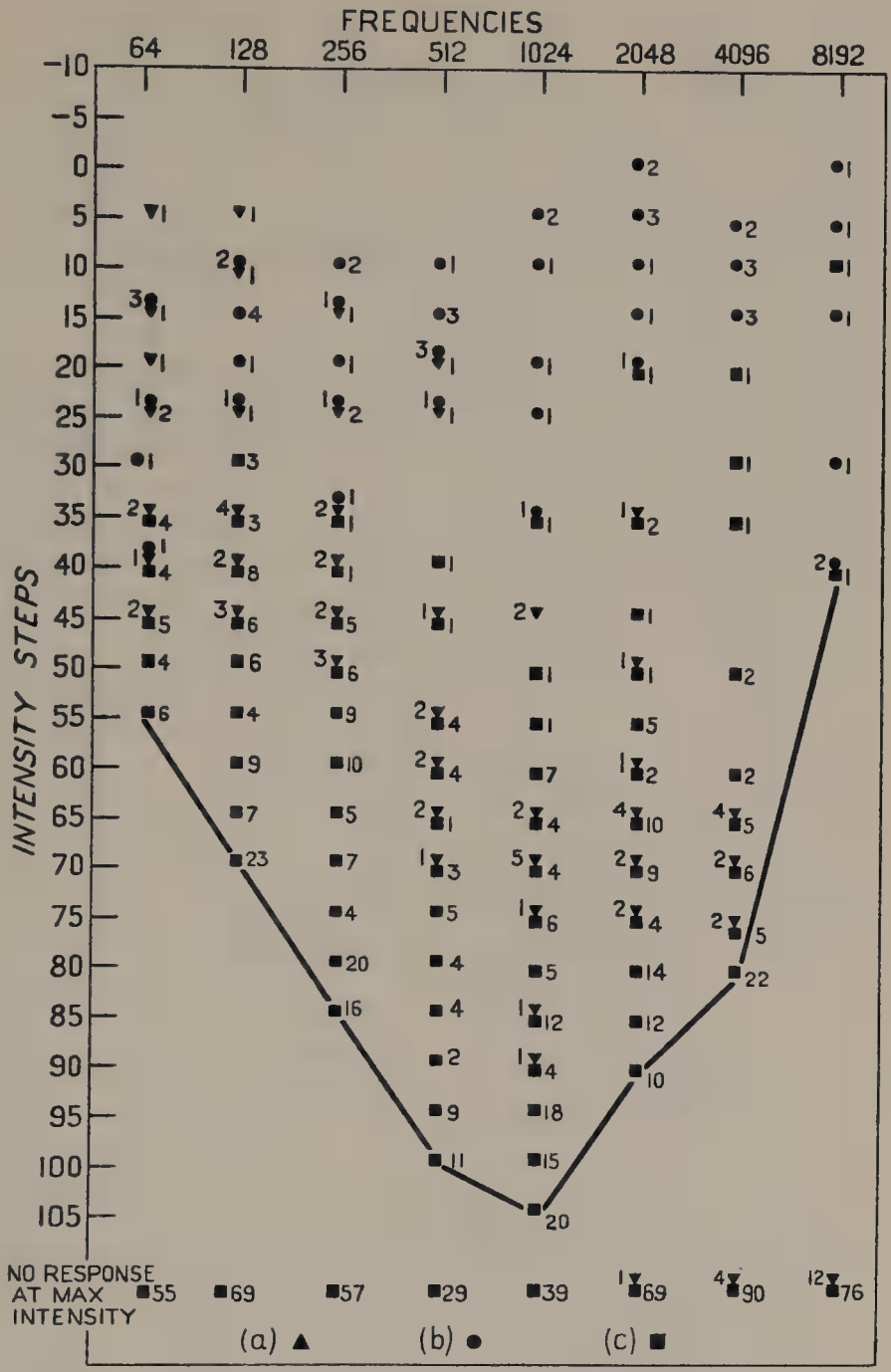
* (Note. No. 208 had normal hearing. He was stated to have lost speech through shock.)

TABLE C. HEARING CAPACITY OF DEAF CHILDREN—*continued*

	64	128	256	512	1,024	2,048	4,096	8,192
No. 222. Boy, 10 yrs.	nil.	65	75	75	80	65	75	nil.
	,,	nil.	70	80	85	70	70	,,
No. 223. Boy, 10 yrs.	,,	60	80	65	80	75	60	,,
	50	55	60	95	85	80	80	,,
No. 224. Boy, 11 yrs.	55	nil.	75	85	85	80	nil.	,,
	55	65	65	75	80	65	75	,,
No. 225. Girl, 11 yrs.	—	nil.	nil.	—	105	nil.	80	—
	—	,,	80	—	105	90	80	—
No. 226. Boy, 12 yrs.	nil.	70	80	100	95	80	nil.	nil.
	45	60	60	75	75	55	75	,,
No. 227. Boy, 12 yrs.	40	50	45	60	70	55	75	,,
	55	60	75	95	nil.	nil.	nil.	,,
No. 228. Boy, 12 yrs.	nil.	70	65	90	95	85	,,	,,
	35	55	70	70	95	50	30	,,
No. 229. Girl, 12 yrs.	nil.	65	85	nil.	nil.	nil.	nil.	,,
	,,	nil.	85	95	90	,,	,,	,,
No. 230. Girl, 12 yrs.	,,	70	80	85	95	85	,,	,,
	,,	nil.	85	85	95	85	,,	,,
No. 231. Girl, 12 yrs.	,,	,,	nil.	nil.	nil.	nil.	,,	,,
	,,	,,	,,	,,	,,	,,	,,	,,
No. 232. Girl, 12 yrs.	,,	,,	,,	,,	,,	,,	,,	,,
	,,	,,	,,	,,	,,	,,	,,	,,
No. 233. Girl, 12 yrs.	,,	,,	,,	,,	,,	,,	,,	,,
	,,	,,	,,	,,	,,	,,	,,	,,
No. 234. Girl, 12 yrs.	—	65	80	—	105	,,	70	—
	—	70	85	—	60	,,	80	—
No. 235. Girl, 12 yrs.	—	nil.	nil.	—	100	,,	nil.	—
	—	70	80	—	105	,,	,,	—
No. 236. Girl, 12 yrs.	—	nil.	80	—	nil.	,,	,,	—
	—	,,	85	—	,,	,,	,,	—
No. 237. Girl, 12 yrs.	—	,,	nil.	—	85	,,	,,	—
	—	70	70	—	105	90	,,	—
No. 238. Girl, 12 yrs.	—	40	60	—	75	55	35	—
	—	55	65	—	60	35	50	—
No. 239. Girl, 12 yrs.	—	nil.	85	—	60	70	80	—
	—	,,	nil.	—	85	65	nil.	—
No. 240. Boy, 13 yrs.	nil.	,,	,,	nil.	105	nil.	,,	nil.
	,,	,,	,,	,,	nil.	,,	,,	,,
No. 241. Boy, 13 yrs.	,,	,,	,,	,,	100	,,	,,	,,
	,,	,,	,,	,,	nil.	,,	,,	,,
No. 242. Girl, 13 yrs.	45	45	60	60	60	70	,,	,,
	50	45	50	60	55	45	,,	,,
No. 243. Girl, 13 yrs.	nil.	70	70	75	65	80	65	40
	,,	nil.	nil.	nil.	100	80	80	nil.
No. 244. Girl, 13 yrs.	40	35	70	80	95	nil.	nil.	,,
	nil.	70	80	100	100	,,	,,	,,
No. 245. Girl, 13 yrs.	,,	70	nil.	100	95	85	,,	,,
	,,	nil.	,,	100	90	80	,,	,,
No. 246. Boy, 13 yrs.	,,	,,	,,	nil.	nil.	nil.	,,	,,
	,,	,,	,,	,,	,,	,,	,,	,,

TABLE C. HEARING CAPACITY OF DEAF CHILDREN—*continued*

	64	128	256	512	1,024	2,048	4,096	8,192
No. 247. Girl, 13 yrs.	—	nil.	nil.	—	100	90	nil.	—
	—	,,	85	—	95	90	,,	—
No. 248. Girl, 13 yrs.	—	45	45	—	nil.	nil.	,,	—
	—	45	50	—	,,	,,	,,	—
No. 249. Girl, 13 yrs.	—	nil.	nil.	—	,,	,,	,,	—
	—	70	65	—	105	,,	,,	—
No. 250. Girl, 13 yrs.	—	50	55	—	85	70	75	—
	—	50	60	—	95	70	80	—
No. 251. Girl, 13 yrs.	—	nil.	nil.	—	90	65	65	—
	—	45	70	—	90	80	65	—
No. 252. Boy, 13 yrs.	—	nil.	85	—	90	70	nil	—
	—	30	35	—	105	80	,,	—
No. 253. Boy, 13 yrs.	—	70	80	—	90	65	80	—
	—	nil.	nil.	—	85	65	80	—
No. 254. Boy, 13 yrs.	—	70	85	—	95	70	nil.	—
	—	40	45	—	75	80	,,	—
No. 255. Boy, 13 yrs.	—	nil.	nil.	—	105	nil.	,,	—
	—	,,	85	—	95	80	,,	—
No. 256. Boy, 14 yrs.	55	60	60	75	95	75	75	nil.
	nil.	60	80	80	85	85	nil.	,,
No. 257. Boy, 14 yrs.	,,	nil.	nil.	nil.	105	85	,,	,,
	,,	,,	,,	,,	105	85	,,	,,
No. 258. Boy, 14 yrs.	,,	,,	,,	,,	nil.	nil.	,,	,,
	,,	70	,,	,,	105	,,	,,	,,
No. 259. Boy, 14 yrs.	,,	nil.	,,	100	100	,,	,,	,,
	,,	,,	,,	100	105	,,	,,	,,
No. 260. Boy, 14 yrs.	,,	,,	,,	95	nil.	,,	,,	,,
	,,	,,	,,	nil.	,,	,,	,,	,,
No. 261. Boy, 14 yrs.	,,	,,	,,	,,	,,	,,	,,	,,
	,,	,,	,,	,,	,,	,,	,,	,,
No. 262. Boy, 14 yrs.	—	70	80	—	100	85	,,	—
	—	70	80	—	85	65	65	—
No. 263. Girl, 14 yrs.	40	40	55	70	100	nil.	nil.	nil.
	35	40	55	70	95	,,	,,	,,
No. 264. Girl, 14 yrs.	—	nil.	85	—	85	80	75	—
	—	,,	75	—	75	60	65	—
No. 265. Girl, 14 yrs.	—	,,	nil.	—	nil.	85	80	—
	—	70	70	—	105	nil.	nil.	—
No. 266. Girl, 14 yrs.	—	nil.	nil.	—	nil.	,,	,,	—
	—	50	45	—	80	,,	,,	—
No. 267. Girl, 14 yrs.	—	65	60	—	85	90	80	—
	—	60	60	—	80	75	75	—
No. 268. Girl, 15 yrs.	—	60	80	—	70	65	50	—
	—	45	55	—	70	60	60	—
No. 269. Girl, 15 yrs.	—	35	55	—	75	70	80	—
	—	55	65	—	85	75	80	—
No. 270. Girl, 15 yrs.	—	nil.	85	—	95	90	80	—
	—	,,	nil.	—	nil.	nil.	80	—
No. 271. Girl, 15 yrs.	—	70	85	—	100	,,	80	—
	—	nil.	nil.	—	105	,,	80	—



Hearing Capacity, tested with the Audiometer and Tunnel Apparatus, of

(*a*) High-Frequency Deaf Children. Table B, p. 40.

(*b*) Linguistically Retarded Children. Table B, p. 40. cf. pp. 142–5.

(*c*) Deaf Children. Table C, pp. 42–4.

(Number of readings at each step is stated, e.g. 5.)

HEARING CAPACITY OF DEAF CHILDREN

The children were unselected. They were tested in two groups, the first 39 in October and November 1928, using the Tunnel Apparatus, the second, 32 in number, in January with the Lamp Signal method of reaction (p. 36). The second group of children happened to be fully capable of understanding instructions given by lip-reading. With the second group the test was shortened by the omission of frequencies 64 and 8192 v.d., which the first group had rarely been able to hear at the maximum steps of intensity, '55' and '40', on the 2A audiometer. Frequency 512 v.d. was also omitted since the aim was to discover the relative hearing for fundamental and partial tones (pp. 55–6). I believe that the results for the two groups may be satisfactorily compared and are not affected by the use of the different methods of reaction.

CAUSES OF DEAFNESS

It would have been valuable to have known which children suffered from congenital and which from acquired deafness, and in cases of acquired deafness the length of time since the hearing first became affected.

The cause of deafness, however, is not known in many instances. Among the patients in Table C the information available is as follows:

born deaf	29
,, partially deaf	1
meningitis	7
measles	1
whooping cough	1
convulsions	1
aphasia (No. 208)	1
nasopharyngeal trouble	1
fall at 7 months	1
concussion	1
middle-ear inflammation	1

leaving 24 patients unaccounted for.

FEW CHILDREN TOTALLY DEAF WHEN TESTED BY THE AUDIOMETER

It was remarkable that only 7 of the deaf children were unable to respond to any sound made by the audiometer,

although the greatest intensities used fell considerably short of the 'threshold of feeling' (p. 26). Many of the children who were tested had never been consciously aware of being able to hear any sound before. In some cases during the testing of the deaf children the sudden lighting up of the face on first hearing the sound was remarkable. This was also noted by Nemzek and Hays (pp. 67–8). They used an audiometer of the same type as ours to test patients in the U.S.A. in 1925–6. The number is not given nor norms quoted, but they published an account of their work, which will be referred to in the next chapter.

The results given in Table C lead to the conclusion, which Nemzek and Hays also expressed, that the audiometer as a means of testing hearing gives quantitative measurements of a range and accuracy unobtainable with any other known form of apparatus. It has made known the existence of hearing in children who had been hitherto considered totally deaf to sound.

TABLE D. HEARING CAPACITY OF DEAF AND NORMAL ADULTS

Frequencies	64	128	256	512	1,024	2,048	4,096	8,192
No. 11	nil.	60	70	80	100	90	nil.	nil.
	50	65	70	75	95	90	„	„
No. 12	No responses.							
No. 13	„							
No. 14	45	50	30	55	60	40	15	nil.
	45	50	50	55	50	50	35	35
No. 15	nil.	60	75	80	70	50	75	nil.
	45	50	55	65	60	70	nil.	„
No. 16	25	20	20	15	15	0	5	5
	10	10	20	25	10	− 10	− 5	5
No. 17	−	− 20	−	− 5	−	10	5	−
	−	25	−	35	−	15	15	−

(− = not tested on these frequencies).

HEARING CAPACITY OF DEAF AND NORMAL ADULTS

The results of testing a few older deaf and normal individuals have also proved useful. No. 11, aged 16 years at the date of testing, suddenly lost his hearing at the age of four

years as the result of a fall. Thanks to proficiency in lip-reading he is now at a public school. I have taught him at intervals since April 1927.

No. 12, aged 16 years, became deaf at $2\frac{1}{2}$ years, after tubercular trouble.

There were three adult patients aged 40–50 years.

No. 13 was born deaf.

Nos. 14 and 15 had a history of otosclerosis and catarrhal deafness extending over more than 10 years.

There were two normal adults, Nos. 16 and 17, aged 17 and 25 respectively.

Nos. 13, 14, 15, 16, and 17 have all been under my observation for several years.

In the next chapter a systematic attempt will be made to evaluate the results of testing this total of 100 individuals, with particular reference to the physical analysis of speech sounds, and the effect of 'high-frequency deafness' on the natural development of speech and language.

CHAPTER IV

HEARING CAPACITY AND SPEECH DEVELOPMENT

(*a*) RELIABILITY OF THE TESTS OF NORMAL CHILDREN (TABLE A.)

THE wide distribution of the readings from step '–20' to step '55', i.e. over a maximum range of 16 steps of intensity, was at once noticeable.

The 'scatter' of these readings is shown in graphic form on p. 37.

Median computed from Table A:

Frequencies	64	128	256	512	1,024	2,048	4,096	8,192
Intensity (nearest step)	25	25	25	30	25	10	10	15

The semi-interquartile range for Table A is shown on p.37.

AUDIOMETER TESTS BY ZUEHL

Zuehl with an Iowa audiometer tested 275 individuals in three groups.

Group I. 25 boys and 25 girls aged 6–15 years.

Group II. 100 males and 100 females chiefly aged 18–23 years.

Group III. 25 sex not given, aged 42–73 years.[1]

It is unfortunate that Group II contained individuals up to 41 years of age. Nevertheless, a clear age difference was obtained.

Zuehl's experiments differed from those here recorded:

(1) He tested each intensity at successive frequencies.[2] I tested each frequency at successive intensities.

(2) He proceeded from 'sound heard' to 'sound not heard'.[3] I found the reverse process much more effective with young children.

(3) The Iowa audiometer (see p. 25) covers a wider range of frequencies than ours, in which the complete speech range, however, is represented.

[1] Zuehl. 89 and 90. [2] Ibid. 88. [3] Ibid.

(4) In the Iowa audiometer intensity steps are marked on a different method from ours, and calculated on an arbitrary basis (p. 25).

(5) His Group I was partially composed of boys and girls from 8–15 years old, of whom I had none among the normal children.

The median for his Group I[1] (calculated from his graph) was:

Frequencies	100	200	500	1,000	2,000	4,000	8,000
Intensities (nearest step)	13	10·5	7·5	3	6	7·5	12·5

with a wide 'scatter'—maximum variation of intensity readings from the median on any frequency not given, but it is more than 8 steps at 4,000 v.d., though less than 2 steps at 2,000 v.d.

The resulting curve, therefore, has a crest at 1,000 v.d. and falls towards each extremity. At the frequencies not included above, because they are outside the tonal range of speech (pp. 55–6 below), the median for Group I drops to approximately the 16th step at 30 v.d.—the lower limit, and at the higher limit 14,500 v.d., to the 18th step. There is a secondary crest with a median of 6 at 3,000 (not given above).[2]

My median curve tends to drop at the extremities, but the crest occurs at 2,048 and extends to 4,096 but not 1,024. In my tests reaction to 2,048 at lower intensities than to 1,024 occurred

in Table A (normal)	20	times out of a possible	24.	
,, B ('aphasic')	11	,,	,,	18 (2 equal readings).
in Table C (deaf)	59	,,	,,	110[3]
,, D (adult)	5	,,	,,	7

This, therefore, is a constant feature. It is not, I think, due to the method of testing. It is improbable that there is a constant difference on this point between American and

[1] Zuehl. 91. [2] Ibid.

[3] i.e. Where figures were available for both frequencies and in spite of the lower maximum intensity of 90 for 2,048 compared with 105 for 1,024.

English children. The more likely suggestion seems to be an unknown difference in the characteristics of the sounds produced by the two audiometers—possibly the feature of 'extensity' of which so little is known, or possibly a mechanical peculiarity.

The age difference between Zuehl's three groups was the superior ability of children to hear notes of frequency above 6,000 v.d. Zuehl's norms for Groups II and III[1] (calculated from his graphs) were:

Frequencies	100	200	500	1,000	2,000	4,000	8,000
Intensities							
(nearest step) II	13·0	10	7·5	4·5	7	9	13
III	13·5	12	9·0	5·0	8	11	17

Amongst his other conclusions[2] were:

(i) That there is less variation in acuity for hearing tones which correspond to the range of the human voice than for any other frequencies.

(ii) That the central value of the composite hearing curves deviate uniformly from a straight line (e.g. the crests indicated): these deviations are not tone gaps or tone islands, but are due to the apparatus and normal factors in the auditory mechanism. (But note my crest at 2,048 discussed above).

(iii) Deviations from the norm are due primarily to differences in the auditory sense organs . . . and secondarily to non-auricular factors . . . e.g. motor, intellectual, emotional, &c.

(iv) A significant hearing inferiority in hearing ability may be present in one or both ears (of the 'normal' individual) without the individual being aware of it.

(v) An inferiority of two intensity steps at points within the frequencies corresponding to the speech range is to be regarded as significant.

(vi) Events in health history have a higher correlation with variations in hearing ability than the latter have with chronological age.

(vii) No significant difference was found between male and female observers of the same age group.

[1] Zuehl. 92–3.

[2] Ibid. 95–6.

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FLETCHER'S AVERAGE NORMAL THRESHOLD OF AUDIBILITY

The apparatus and methods by means of which Fletcher calculated the average threshold of audibility for about 100 American men and women, free from auditory defects, have been outlined in Chapter II.

The step marked '0' on the No. 2A audiometer used in this research represents this average threshold.[1] It is necessary to ask why my median departs from it.

Briefly, there appear to be three possible reasons.

First, the fact that tests by Fletcher and associated workers were carried out in sound-proof booths while mine were made in an office in a school building, where the masking effect of extraneous noise could not be altogether avoided (e.g. p. 28). Secondly, I tested children, aged 6 to 8 years, while Fletcher tested adults. (It should be noted that Zuehl's Group I contained boys and girls up to 15 years of age).[2]

From the present research it appears probable that young children are not so sensitive to pure tones as older boys and girls or adults.

There is good evidence for believing that young children are not easily interested in them and that therefore attention is less strong.

There is also a third possible reason, that the normal children whom I tested might have been able to hear rather lower intensities of the notes used if there had been a longer pause at each intensity step. Smith and Bartlett, in a series of tests with a buzzer noise as stimulus, found that 5 seconds sometimes elapsed before an individual was aware of the noise at the threshold of audibility.[3] Such protracted tests, over the whole frequency range of speech, are not possible with young children.

(*b*) THE PHYSICAL ANALYSIS OF SPEECH SOUNDS

It will be shown by means of a summary of the data obtained by the principal modern workers that frequency-differences are the essential characteristic of the physical

[1] Fletcher, H., 216. [2] p. 49. [3] Smith and Bartlett, 117.

nature of speech sounds. In the next part of the chapter evidence will be given that this fact is of the greatest consequence in relation to the development of speech and language in children who are partially deaf, whether the trouble is 'high-frequency deafness' or of some other type.

1. BY MILLER

In 1917 Miller published a full account[1] of his important and thorough investigation of the sounds of human speech by the methods of pure physics. He had invented an apparatus to which he gave the name 'phonodeik'.[2] Sound is collected by a resonator horn of known natural period and directed by it on to a glass diaphragm 0·003 in. thick. 'Behind the diaphragm is a minute steel spindle mounted in jewelled bearings, to which is attached a tiny mirror; one part of the spindle is fashioned into a small pulley; a few silk fibres or a platinum wire 0·0005 in. in diameter are attached to the centre of the diaphragm and being wrapped once around the pulley are fastened to a spring tension piece; light from a pinhole is focussed by a lens and reflected by the mirror to a moving film . . . in a special camera. The vibrations of the diaphragm, consequently, are transformed into movements of a beam of light which are recorded on a photographic film.'

The resulting records are analysed by Fourier's Theorem,[3] with the help of Henrici's Harmonic Analyser.[4] A verification is then made by harmonic synthesis using a 32-component harmonic synthesizer.[5]

Miller investigated noises [6] and musical sounds [7] as well as those of speech. He corrected errors due to the inherent features of the apparatus.

By these methods he analysed 8 English (American) vowels, at several pitches, as intoned by each of 8 voices giving about a thousand curves.[8] The voices were 2 basses, 2 baritones, a tenor, a contralto, a boy soprano (14 yrs.) and a girl soprano (10 yrs.). He also took 202 [9] photographs in a definitive investigation of one voice.

He concluded that 'all vowels may be divided into two

[1] See bibliography. [2] Miller, 78–83. [3] Ibid. 92.
[4] Ibid. 98. [5] Ibid. 113. [6] Ibid. 141.
[7] Ibid. 175–214. [8] Ibid. 221–31. [9] Ibid. 224.

classes, the first having a single, simple characteristic region of resonance, while for the second there are two characteristic regions.'[1] (For results see summarized Table E, below, p. 56).

2. BY STUMPF

Miller's figures are confirmed by those of Stumpf,[2] in spite of a different language—German—being used by the latter and a wholly different method. 'Stumpf's analysis was made with the aid of interference-tubes carefully adjusted with regard to all the partials of the fundamental tone upon which the vowel was being spoken or sung. His apparatus permitted him to include or exclude at will the various partials of the voice-tone. He was thus able to determine just which partials were characteristic of the vowel, and which ones were accessory or irrelevant. After determining the formants in this manner, Stumpf performed a painstaking synthetic experiment in which pure tones were assembled in the number and intensity necessary for the production of various vowels. The outcome of these syntheses was successful . . .'[3]

A number of subjects were used in these experiments.

3. BY PAGET

Paget analysed English vowels and consonants spoken and sung by himself. His methods included forcing breath through the mouth without vibrating the vocal chords, and 'sounding' the cavity of the pharynx, while the jaw, tongue, lips, and soft palate were adjusted as if to utter vowels and consonants.[4]

4. BY CRANDALL

The most complete analysis of the sounds of speech of which records are published was made by the Staff of Bell Telephone Laboratories (U.S.A.) under the direction of Crandall.[5]

'Briefly stated, the principles involved in this method are as follows: Speech waves are picked up by a telephone transmitter and converted into electrical waves. They are then

[1] Miller, 225. [2] Ogden, 81–102. [3] Ibid. 84–5.
[4] Paget. See bibliography. [5] Fletcher, H., 26.

magnified by means of a special amplifier and sent into an oscillograph, where they cause a tiny ribbon to vibrate. The motion of this ribbon is then photographed on a moving film.'[1] The whole and each part of this apparatus were calibrated by exact methods and the conclusion was that it was nearly free from distortion.[2] 'The apparatus was sufficiently powerful to record sounds spoken in an ordinary tone of voice when the speaker's lips were about 3 inches from the transmitter.'[3] The photographic records were analysed.

Crandall concluded that the general characteristics of speech were as follows:

LARYNGEAL OR FUNDAMENTAL PITCH

'The pitch of the voice when speaking the vowels varies with different individuals, corresponding to about 90 cycles (double-vibrations) per second for a very deep-voiced man and to about 300 cycles per second for a high shrill-voiced woman. The average pitch used by a woman is near Middle C or 256 cycles per second.'[4]

VOWEL CHARACTERISTICS

Together with the laryngeal or fundamental tones are produced, during the utterance of vowels, vibrations of higher frequency which may be described as harmonics or partials.[5] These give the characteristic quality to each vowel and are brought about by adjustments of the jaw, tongue, and lips.[6] Like the earlier workers mentioned Crandall found that there are two groups of vowels.[7] In the first group there is a strong lower frequency characteristic, but there is also a higher frequency characteristic which is small in amplitude. Miller, with his apparatus, found only the lower frequency characteristic for this class of vowel. In the second group both low and high frequency characteristics are important.

Fletcher gives the following table based on the combined results of Miller, Stumpf, Paget, and Crandall.

[1] Fletcher, H., 26. [2] Ibid. 27. [3] Ibid.
[4] Ibid. 53. [5] Ibid. 55. [6] Ibid. 7.
[7] Ibid. 57.

TABLE E. CHARACTERISTIC FREQUENCY OF THE VOWEL SOUNDS[1]

Speech Sound.	*Low Frequency.*	*High Frequency.*
ū (pool)	400	800
u (put)	475	1,000
ō (tone)	500	850
ȯ (talk)	600	950
o (ton)	700	1,150
a (father)	825	1,200
a (tap)	750	1,800
e (ten)	550	1,900
er (pert)	500	1,500
ā (tape)	550	2,100
i (tip)	450	2,200
ē (team)	375	2,400

It will be shown that in 'high-frequency deafness' the distinction between vowels of the first group referred to above, such as ū (pool) and ō (tone) and those of the second group, as ā (tape) and ē (team), is of primary importance. If the high frequency characteristics cannot be heard, ū and ē, ō and ā (and similar pairs of vowels one belonging to each group) sound alike (p. 64).

CONSONANT CHARACTERISTICS

The essential characteristics of the consonants, as of the vowels, have been found by Miller, Stumpf, Paget, and Crandall to be vibrations in particular regions of frequency,[2] The consonants are more complex and 'noisy' (in frequency not intensity) than the vowels. The highest frequency in any consonant is 8,000 v.d. in 'S'.[3]

THE INTENSITY OF SPEECH

In Bell Telephone Laboratories tables valuable to otologists and others have been compiled from the measurement of speech power in terms of the electrical unit, the microwatt.[4]

Vowels were found to involve much greater intensities than the consonants.

'The most powerful sound is ó (awl) and the faintest sound th (thin) the ratio of powers between these two being 680.'[5]

[1] Fletcher, H., 58. [2] Ibid. 59–63. [3] Ibid. 60, 62.
[4] Ibid. Chap. III. [5] Fletcher, H., 72.

'A vowel in an accented syllable has usually three or four times as much phonetic power (maximum value of the mean speech power of the vowel) as one in an unaccented syllable.'[1]

THE FREQUENCY BASIS OF SPEECH SOUNDS

It follows from this that the audible sounds of human speech fall under two groups on a frequency basis:

1. *The Laryngeal or Fundamental Tones—the Pitch of the Voice.* These are the notes produced by the vocal chords, the notes read from the musical score in singing. Experiments show that they vary more rapidly in speech than in singing.[2] In human speech the laryngeal tone conveys emotional changes (discussed by de Laguna, p. 5). The confident voice is characterized by the use of the individual's lower range of laryngeal pitch, emotional stress is reflected in a raising of pitch. This form of vocal expression comes first in the individual development of the human infant; reactions to laryngeal tones precede reactions to words. Variations of laryngeal tone are sometimes stereotyped into patterns, e.g. the raising of the voice in a question.

2. *Vowel and Consonant Characteristic Frequencies.* The physical nature of human speech thus rests primarily on a system of difference in frequencies. But the amount of energy reaching the ear at a given frequency or frequencies plays a great part in determining the auditory experience and the reaction of the individual.

(c) DEAFNESS TO HIGHER VIBRATION FREQUENCIES AND THE ANALYSIS OF SPEECH SOUNDS

In Chapter I it was shown that the natural development of the speech-language function is dependent on the capacity to hear. In the present research the discovery of 'high-frequency deafness' in patients Nos. 1–6, taken in conjunction with the analysis of speech suggested a cause for their apparent aphasia.

The position was as follows:

(i) It was clear that in order to hear and understand speech normally the young child must receive auditory stimulation over a range of frequencies from about 90 (laryngeal tone, bass voice) to about 8,000 (highest frequency for 'S').

[1] Fletcher, H., 74. [2] Miller, 223, and Fletcher, H., 54–5.

(ii) The 'high-frequency deaf' patients were sensitive to frequencies 64 to 1,024 in all cases, 64 to 4,096 in 4 out of the 6 cases, and the constant appearance of the curve for all the readings, backed by results from subsequent testing suggested that if sufficient steps in intensity had been available, sensitivity to all frequencies up to 8,000 might have been found.

(iii) The crux of the problem therefore appeared to be, what degree of acuity is adequate for speech-language development under normal conditions of life?

As regards laryngeal tones of voice, for which Crandall's range 90–300 v.d. is probably sufficiently representative, the acuity of the high-frequency deaf children appeared adequate. The greatest 'loss', represented by the intensity reading 50, fell within the normal distribution (Table A). The essential fact about the normal children was that there was no suspicion or symptom of imperfect speech-language development among them.

The laryngeal tones of the voices of the high-frequency deaf children, with the exception perhaps of No. 4, were normal. The apparent emotional over-excitability noted in patients Nos. 1 and 2 may be partly due to their normal hearing for laryngeal tones. They received in early life and again during special teaching, emotional stimulation evoking social and most ancient reactions in the nervous system (see de Laguna, p. 5) but they were deprived of the inhibiting influence, also social, normally effective through verbal explanation and conversation. The apparent discrepancy in the diagnoses quoted in Chapter I is explicable if it is remembered that not only are the routine tests largely dependent for their efficiency on verbal comprehension and careful judgement on the part of the patient, but that pupil-dilatation in response to 256 would be normal, and that this fork is the one most used. The animal noises imitated by No. 1 were again fundamental and laryngeal notes for which his acuity was normal. When No. 1 recognized the sound of a motor-car engine[1] and a ship's fog-horn, the latter at a great distance, by ear alone, it

[1] In the first month of special teaching he showed by cries and gesture that he had recognized the sound of a car engine being raced, about 40 ft. away, out of sight.

was because the characteristic frequencies of the notes fell within the frequency range whose upper limit was about 256 v.d.

But for the frequencies giving the vowel and consonant differences of speech the high-frequency deaf patients had insufficient acuity to receive adequate stimulation. This condition would also affect reaction to non-speech sounds of the same frequency range. Table E shows that the frequency 512 available with our audiometer approximately represents the lower limit of this range—the upper being approximately our audiometer's 8,192—the region 2,400–8,000 being essential to consonants only.

The effect of the lack of acuity from 512 v.d. was clearly shown before teaching by special methods began. Except as a means of emotional stimulation speech heard was a source of confusion. This applied to a large extent to noises also, which are often characterized by groups of partials, of frequency above 512 v.d. It is extremely probable that by the time teaching began in each case (from $4\frac{1}{2}$ years onwards), a certain indifference to sound, unconscious rather than conscious, had begun to develop.

EFFECT OF HIGH-FREQUENCY DEAFNESS ON THE RECOGNITION OF NOISES

Evidence for the conclusion just stated was obtained by a series of original experiments in the recognition of noises carried out in 1927. Twenty normal children were tested, at St. Chrysostom's Schools, Victoria Park, Manchester. Their ages were from 3 to 7. A high-pitched small bell, a squeaking toy like a puppy, a rather low-pitched whistle, a toy motor-horn, and a low-pitched fairly large Swiss cow-bell were shown to each child, in that order, and sounded. The child's back was then turned and the toys were sounded in a different order—large bell, puppy, horn, small bell, whistle, the child being asked to turn and point to the toy just sounded. Every effort was made to equalize the duration and intensity of the sounds. The arrangement of the toys was exactly the same throughout the experiment, and the child under examination was not allowed to turn round until the toy just used had been replaced. The distance of the stimulus from the patient was kept as far as possible constant throughout the tests—about 3 ft. from the child's head.

The errors of this group of twenty 'normal' children were:

(i) large bell recognized as small bell by

1 out of 4 seven-year-olds.
1 „ 4 five-year-olds.

(ii) small bell recognized as large bell by

1 out of 4 seven-year-olds.
1 „ 5 six-year-olds.
1 „ 4 five-year-olds.
2 „ 3 four-year-olds.
2 „ 4 three-year-olds.

(iii) Two of the six-year-olds also identified the whistle as the horn.

The total number of errors among the normal children was 11 per cent.

Under the same conditions, Nos. 1 and 2 gave:

Stimuli.		L-bell.	Puppy.	Horn.	S-bell.	Whistle.
Reactions:	No. 1.	S-bell.	„	„	L-bell.	„
	No. 2.	Horn.	„	Whistle.	S-bell.	Horn.

Total errors: 60 per cent.

A week later the results were:

Stimuli.	L-bell.	Puppy.	Horn.	S-bell.	Whistle.
No. 1.	Horn.	L-bell.	Puppy.	„	Horn.
No. 2.	S-bell.	Puppy.	Horn.	Puppy.	„

Increasing the distance and varying the order of the stimuli the results were:

(*a*) at double the distance—6 ft.:

Stimuli.	L-bell.	Puppy.	Horn.	S-bell.	Whistle.
No. 1.	Horn.	'Can't hear'.	„	„	Horn.
No. 2.	Puppy.	Puppy.	„	?	Horn.

(*b*) at 12 ft.:

No. 1.	Whistle.	'Can't hear'.	Horn.	S-bell.	Horn.
No. 2.	Puppy.	Horn.	„	Puppy.	Whistle.

Nos. 3, 5, and 18 were tested at the 3 ft. distance. No. 4 was not available:

Stimuli.	L-bell.	Puppy.	Horn.	S-bell.	Whistle.
No. 3.	S-bell.	„	„	L-bell.	„
No. 5.	L-bell.	„	„	S-bell.	L-bell.
No. 18.	„	„	L-bell	„	Whistle.

No. 18 gave the same responses when the stimuli were repeated in a different order at the distance of 6 ft.

The loudest sound, the motor-horn, was most often correctly recognized by Nos. 1 and 2. The two bells were confused by the 'normal' children in 9 instances, as well as by the high-frequency deaf. Whistle and horn were confused by Nos. 1 and 2, also by two of the normal children.

This series of experiments was made before the audiometer tests were initiated, and therefore before I knew of the 'high-frequency deafness' of patients Nos. 1–6, amongst whom Nos. 1 and 2 were most severely affected. Much subsequent day-to-day testing has confirmed the existence of this great difficulty for them in hearing noises.

ACCURATE RECOGNITION OF SOUNDS FULLY HEARD

But if the sound is made of adequate intensity to counterbalance their defect the high-frequency deaf have no difficulty in distinguishing, remembering, recognizing, and interpreting it, as the following original test given in 1928 showed.

TEST FOR THE RECOGNITION OF SOUNDS DIFFERING IN FREQUENCY

THE MOTOR-CAR TEST

Objects—to test:

(*a*) discrimination between, retention and recognition of, sounds of like intensities but unlike pitch;

(*b*) capacity for forming auditory-visual associations.

Apparatus: audiometer, first visible then concealed. Pictures of motor-car, motor-boat, liner, traction-engine, second motor-car, and aeroplane.

Preparatory practice: Patient's interest is sought in audiometer. Patient then seated. Audiometer concealed by screen or behind patient's back. Pictures of first motor-car and motor-boat presented three times in order 1, 2, 1, 1, 2, 2. Each picture presented for 8 seconds and while patient is directed to look at picture he listens to note produced by audiometer. Throughout the test the better ear, right or left, as shown by the results of 'Tunnel Test' previously given, is used. Intensity of frequencies used is 10 per cent. (two steps) above the accepted reading for each frequency

obtained by 'Tunnel Test'. Frequencies for first motor-car and motor-boat pictures used in preparatory practice are 256 v.d. and 1,024 v.d. respectively. Both pictures are then placed before patient. The sounds are produced in order 1, 2, 1, 2, 2, for 8 seconds as before. Patient is asked to indicate correct picture. If patient fails to respond, practice is repeated. Help given in this practice to be noted.

The Test:

Learning process. Procedure similar to that for practice above.

Pictures.	*Sound-frequencies.*
1. Liner	64
2. Traction-engine	512
3. 2nd Motor-car	128
4. Aeroplane	2,048

(Compare these frequencies with characteristic vowel frequencies, p. 56).

Order of presentation: 1, 2, 3, 4, 1, 3, 2, 4, 3, 1, 4, 2.

Recognition process: Four pictures are all presented in arrangement:

1 2
4 3

on the table.

Sounds are produced in order 2, 1, 3, 2, 4, 1, 3, 4, patient being asked to indicate—by pointing—'Which one was that?'

Right or wrong answers are recorded.

TABLE F. RESULTS OF MOTOR-CAR TEST FOR THE RECOGNITION OF SOUNDS DIFFERING IN FREQUENCY

Frequency order: / = correct answer.	2.	1.	3.	2.	4.	1.	3.	4.
No. 1. Right ear. Answers	4	/	/	/	/	/	4	3
No. 2. „ „ „	/	/	/	/	/	/	1/3	/
No. 3. „ „ „	4	/	/	4	2	/	/	/
No. 4. „ „ „	/	/	/	/	/	/	/	/
No. 5. Left „ „	?	/	/	/	/	/	/	/

No. 1's answers when the test was repeated on the next day, were all correct.

No. 2 corrected herself at the second repetition of 3.

No. 3's confusion of 2 with 4 in three instances appeared to be an error in associating picture and sound, since his five other answers were correct.

In view of these results, which indicated no difficulty in distinguishing frequencies—provided the intensities used were adequate—among the high-frequency deaf children, the test was not standardized with normal children and it was thought unnecessary to give it to No. 6 when he was later discovered to be in the same condition (p. 39).

EDUCATION OF HIGH-FREQUENCY DEAF CHILDREN IN LISTENING TO SPEECH

Equally strong evidence that the symptoms of the high-frequency deaf children before special teaching—apparently variable reaction to noises, non-comprehension of speech, and mutism—were due to their subnormal acuity to the speech-partial frequencies above 512 v.d., was provided by an analysis of their speech-language development in response to specialized methods of education. At first it was only possible to develop the beginnings of speech and language among these patients with the use of the technique employed in teaching deaf children—using sight and touch. But from the earliest lessons onwards every effort was made to teach the patients to listen to speech. In 1929, No. 1 was the most advanced of them in this respect—he had had 5 years of concentrated individual attention for an hour or more daily—and could acquire new and difficult words by ear alone.

(1) *Vowel confusion.* Special difficulty in acquiring *exact* normal English vowels was shown by Nos. 1 and 2. They learned to utter them so nearly correctly that even a 'narrow' system of phonetic symbols was inadequate to show the fine differences in quality between their pronunciation and that of their social environment.[1] But there were also direct confusions of completely different vowels, inexplicable before the audiometer tests in 1928. In February 1927 a striking instance occurred with patient No. 1. I was giving him a learning test. He was required to learn a verse of poetry by ear alone, sitting with his back turned and repeating the words, line by line, then a verse at a time after me.

[1] With further teaching this difficulty has disappeared.

I said: 'Where he comes from nobody knows, Or where he goes to, but on he goes.'

He repeated correctly to 'goes to' but continued 'but on his way'. This on two successive days, a third attempt being 'where he goes to no one *knows*'.

Accordingly, I tested Nos. 1 and 2 by methods similar to those of the 'Motor-car Test' above, using the vowels m*ow*, m*a*te, m*oo*, and m*ee*. Other vowel sounds were often included in these tests but the four just mentioned proved the most frequent sources of error. Tables B and E taken in conjunction seem to give a clear explanation of the cause.

Using methods similar to those of the 'Motor-car Test' I investigated the occurrence of this phenomenon among the high-frequency deaf children when spoken numbers were the stimuli. The intensity of the speech used was regulated as accurately as possible, but the main feature throughout was the frequent occurrence of certain confusions when the distance between patient and speaker was increased or the intensity of the sounds deliberately lowered.

Nos. 1 and 2 were given practice from September 1927 onwards in discriminating between vowels, and they have been able to distinguish them at progressively greater distances and lower intensities, but the difficulty still exists if the intensity is for them inadequate.

No. 1, in a series of recorded practices, following the method described above, repeatedly confused 'two', 'three', and 'four' (see analyses, Table E). In 50 possible chances of confusing these words there were 27 correct answers, 11 instances of confusion, and 12 where the patient was doubtful.

No. 2 responded in the same way.

No. 3—with less than 2 years at school, under the same conditions as No. 1 and with the same numbers—made 5 right, 2 wrong, and 6 doubtful answers in 14 chances.

No. 4, after 5 years at school made 21 right, 3 wrong, and 2 doubtful answers, in 26 chances.

No. 5, after 10 years at school, and with the best acuity to high frequencies of the group, made 15 right, 2 wrong, and no doubtful answers in 17 chances.

No. 6, after 7 years at school, made 12 right, 2 wrong, and no doubtful answers in 14 chances.

It is noticeable that No. 202 with a similar record of loss for high frequencies made no errors or doubtful answers in 14 chances. He proved to have become deaf after an attack of measles at the age of 5 years, i.e. after speech and language had been developed and firmly established.

(2) *Consonant confusion.* Patients Nos. 1 and 2 have had difficulty in distinguishing between syllables with like vowels but unlike consonants. It would be possible to make a very long list of such errors. The examples given are confusions in words heard. No. 1, in 1925, thought I said 'door' when I actually said 'drawer', he confused 'candle' and 'handle', 'hole' and 'coal', 'glass' and 'brass', 'stamp' and 'camel', he said 'commerhouse' for 'summerhouse', 'bread' for 'spread'. In 1927, in dictation, I said '. . . tunnel', he said 'Funnel?', I said '. . . dashed into the water', he said '. . . splashed into the water'. I said '. . . took . . .' in a sentence, he repeated '. . . put . . .'; I said 'club' and he said 'glove'. No. 2, in 1925, had very little special teaching. She developed a jargon, e.g. 'pukky, upkay' = 'put it upstairs', 'kaybi' = 'table', 'nuff' = 'glove', 'meemi hairful' = 'do be careful'. In 1927 I said 'fog', she questioned 'frog?' and then 'frost?'. She confused 'caught' and 'talked', 'handle' and 'candle'; in 1928 'candle' and 'tunnel', 'bought' and 'brought'; in 1929 'tram' and 'sand'.

I find evidence that Nos. 1 and 2 have acquired the consonants in speech through lip-reading and touch, through the special methods used for teaching speech and reading. (See Part II, Chapter VI, pp. 115–18.) In March 1929 they could distinguish by ear alone vowels spoken in an ordinary voice at 6–10 ft., but consonants only at 2 ft. at the most. In speech they were only then beginning to use a correctly articulated 's' in spite of continuous attention and help. 'S' is characterized by the highest region of partials in speech, extending up to about 8,000 v.d. (p. 56).

Nos. 3, 4, and 6 obviously have considerable difficulties with the consonants in speech, and with No. 5 I have noticed the substitution of 'k' for 't' in words.

(3) *Unstressed or unemphasized syllables not heard.* These carry less intensity and energy (see p. 57), and I have noted difficulty in hearing them among the high-frequency deaf

patients, e.g. No. 1, in 1927, repeating after me for 'This caravan has windows two' said 'This caravan *got* windows two'; for 'He called out in an angry tone', said 'He call in angry tone'.

No. 2, in 1929—also repeating after me—for 'We wanted to buy some potatoes' said 'We wanted some potatoes', and for 'the tram ran into a bus' said 'The tram were one into a bus'. For 'the windows of the tram were broken', she said 'the window of the tram was broken'.

These instances are typical of those which occurred daily; one feature characterizes them all, the errors occurred on the unstressed or unemphasized syllables or words, that is to say unstressed as said by me on the occasions in question.

MINIMUM HEARING NEEDED FOR NORMAL SPEECH DEVELOPMENT

The question arises as to what degree of auditory acuity over the vowel and consonant frequency range[1] is necessary for the intensities of normal speech to prove an adequate stimulus to normal and natural speech-language development in the young child. The evidence on this point is not as clear as for the fundamental range (128–256 v.d. on our audiometer). Table A (Normal Children) shows

1	reading at	45	'loss'
6	readings at	40	,,
6	,,	35	,,
and 11	,,	30	,,

that is to say, 24 of the total number of 120 readings for the 5 frequencies 512, 1,024, 2,048, 4,096, and 8,192, or 20 per cent. occur at intensity steps above 25 on the 'loss of hearing' dial. And none of these children showed any defect in speech-language development.

Among the children at the Royal Schools for the Deaf (Table C), whose deafness is definitely so severe as to interfere with speech-language development and maintenance under normal conditions of life, there were two patients, Nos. 213 and 202 (p. 65) with three or more readings less than 45 on the frequency range 512–8,192. Apart from this there were

[1] i.e. 512–8,192 as represented on our audiometer.

only 8 isolated readings of 45 or under for the vowel and consonant frequency range among the remaining 66 patients. It is highly probable, therefore, that readings of about 45 represent the border-line between acuity adequate for speech-language development and acuity which is inadequate and necessitates special methods of teaching. The evidence of patients Nos. 14 and 15 suggests that the line of demarcation should be placed if anything at a lower step of intensity.

(*d*) AUDIOMETER TESTS AS A MEANS OF DIAGNOSIS

Bunch, using an Iowa Audiometer of different model to Zuehl's,[1] and Nemzek and Hays[2] with a Western Electric Audiometer of the same pattern as our own, tested patients suffering from various forms of ear trouble. The total number of patients is not stated in either case. Bunch, like Zuehl, proceeded from 'sound heard' to 'sound not heard' and varied the frequencies at a given intensity (pp. 33 and 49).

Nemzek and Hays also proceeded from 'sound heard' to 'sound not heard'; they do not say whether they examined with fixed intensities or fixed frequencies.

In drawing conclusions from the two series of experiments these three workers agree in stating that audiometer tests are of great value to the physician for purposes of diagnosis. It was to elucidate this point that Nemzek and Hays, themselves consulting physicians, undertook the inquiry. Bunch collaborated with Dr. Dean of the Otological Clinic at Iowa.[3]

Bunch presents records of three patients suffering from deafness originating in the external ear, also two records illustrating the effect of packing the external canal of a 'normal' adult with boric acid and powder. In the latter instance there is 'a uniform lowering of the curve throughout the entire range'.[4]

Two records of 'pure middle ear lesions' (Dr. Dean) are given, due to acute tubal catarrh and an 'adenoma in the fossa of Rosenmüller, in the right side causing a mechanical blocking of the eustachian tube' respectively.[5] Five patients

[1] Bunch. See bibliography.
[2] See bibliography.
[3] Bunch, 45.
[4] Ibid. 70–1.
[5] Ibid. 72–4.

suffering from combined middle-ear and inner-ear troubles, three whose deafness was due to acoustic neuritis, two to neuro-labyrinthitis, and two to otosclerosis are discussed.[1]

Bunch found that it was impossible to determine a 'normal curve' for various diseases 'because the relative development of the disease would determine the general contour of the curve given'.[2] I attempted to correlate readings in Table C with the cause of deafness, without success.

A careful examination of all these records shows that while there was a uniform lowering of acuity over the whole frequency range for speech in purely external ear troubles (medicated cotton not removed from the ear for 18 months), and in the pure middle-ear lesions (above), the five acoustic neuritis and neuro-labyrinthitis cases without exception show normal or relatively normal hearing for lower frequencies and marked and severe loss for higher frequencies. In otosclerosis there was marked loss throughout the entire range.

These findings are of great importance in the present research where the high-frequency deaf patients (Table B) all showed severe loss of acuity on the higher frequencies with normal or relatively normal hearing for the lower. There is, however, a point of difference between four of these patients and those described by Bunch: Nos. 1, 2, 3, and 4 all showed marked loss from 512 v.d. upwards, Bunch's from 770 v.d as the lowest frequency.

Bunch states the 'audiometer has been materially helpful in differentiating between diseases of the inner and the middle ear'.[3]

Nemzek and Hays, in their shorter account, give fewer details of their work, but the following quotations are important: 'We find in most cases (of middle-ear inflammation) along with loss of hearing for the lower notes we usually have loss of hearing for the higher notes and in most cases complete loss after 4,096 v.d. We attribute this phenomenon to congestion of the inner ear concurrently with the middle-ear inflammation. The higher notes often return to normal quite rapidly after proper drainage is instituted.'[4]

[1] Bunch, 74–9.
[2] Ibid. 69.
[3] Ibid. 80.
[4] Nemzek and Hays, 568.

AETIOLOGY OF HIGH-FREQUENCY DEAFNESS

As regards the high-frequency deaf patients with whom this research is primarily concerned, the reports of the aural examinations made by Dr. H. C. Smith (pp. 149–50) show some lack of uniformity as contrasted with the uniform features of the loss of auditory acuity, namely slight differences between the two ears (one instance of a difference of 5 steps—with No. 2 at 2,048 v.d. as against several greater differences among the normal children, Table A, p. 38), and steady increase of loss of acuity from frequency to frequency, from 128 v.d. upwards, over the speech range.

Nos. 1 and 2 present uncomplicated pictures, without history of disease or injury. With No. 5 alone, of the high-frequency deaf children, was a cause of deafness suggested in a previous diagnosis. On this boy's 'admission form', completed when he was sent to the school for the deaf, meningitis at 4 years was recorded. Dr. H. C. Smith, however, considers that the condition is probably congenital (p. 149).

With Nos. 1 and 2, at any rate, middle-ear, conductive defects seem to be clearly and absolutely ruled out. Even with No. 3 it is Dr. Smith's opinion that the middle-ear disorganization which he found is not sufficient to account for the severe loss of acuity from 512 v.d. upwards.

Further than this, in the present state of knowledge, it is impossible to go (cp. criticisms by Somerville Hastings and Clarke, pp. 17–19).

A defect in the perceptive apparatus is indicated. But the site of this defect, whether in the cochlea, or at one or more levels of the auditory path, or in the primary auditory centres of the brain, cannot be localized.

The investigations of Head[1] show that there may be groupings and regroupings of the nervous impulses which leave the cochlear nuclei before they give rise to the relatively simple sensations which we associate with sound. It is not known whether the half-way station on the auditory path, at the internal geniculate body, has any sorting or sifting function of this kind.

It is also worth while to remember that the sense of hearing

[1] *Studies in Neurology*, 1920.

is the last to develop in the life of the individual and that myelination of the auditory path occurs later than with the other sensory paths.

Is it possible that the condition of Nos. 1 and 2 and the other high-frequency deaf patients is mainly due to retarded development in this respect?

Such an inquiry is outside the scope of this research, and in fact the technique for it has not yet been developed by any worker.

(*e*) THE RESULTS OF TEACHING BY SPECIAL METHODS

(cf. pp. 115–18 and 128–31)

In Chapter I a description was given of the apparent deafness, except at infrequent intervals, the non-comprehension of speech and language and the mutism of high-frequency deaf patients before special teaching.

The following specimens of conversation illustrate the results of this teaching.

No. 1, 1929, after 5 years' continuous teaching. 'I'd like to be able to photograph a liner. I may have a camera soon . . . I've got four photographs of myself.' 'I fitted a little electric light to it, you press a little switch and it goes on.'

No. 2, 1929 (continuous special teaching since June 1927, —before that, though begun in 1922 it was at irregular intervals): 'That (=that's) a new book from Donald.' 'Mother and Daddy gave me my birthday some nice clock.' 'Martyn and I went out on ship with Uncle Jim.'

These children can converse freely on any topic suitable to their age. The full extent of their development is not represented in any way adequately by brief quotations. It is analysed much more fully in Part II, Chapter VI, and the striking results of some of Burt's Scholastic Tests given.

POSSIBILITY OF IMPROVEMENT IN POSITIVE AUDITORY ACUITY OF HIGH-FREQUENCY DEAF CHILDREN

There is considerable controversy as to whether in pathological conditions of the auditory apparatus, it is possible to bring about improvement in positive auditory acuity as the result of training. Usually such training takes the form of causing the patient to listen to certain loud, mechanically

produced sounds.[1] The treatment is given for fixed periods of time, at frequent intervals. Marked improvement, with a number of patients, is stated to have been obtained. An official report of the United States Bureau of Standards, however, included a general pronouncement against such methods, though it is clear that treatment by medical men and on scientific principles had not been considered.[2]

In the present research it was decided to investigate this problem on a small scale in so far as it affected the high-frequency deaf children Nos. 1 and 2. These children were having daily practice in listening to the sounds of loud speech. There was no doubt that whereas they were completely unable to understand speech before their education by special methods, they were now able to follow conversation under certain conditions by hearing alone (pp. 63–6 and 115–18). Nos. 1 and 2 can now as a rule follow the conversation of other children under normal conditions. It is possible to talk to No. 1, while his back is turned, in an ordinary conversational voice, at a short distance of 1–2 ft., and be understood perfectly. Had their positive auditory acuity improved through the special methods of education and training, or was their progress in hearing due solely to their having learned to make use of the limited hearing which they possessed?

It will be seen that, at present, there is no certain answer to this question. But Smith and Bartlett (p. 52) noted that after practice the adults whose auditory acuity they tested were able to hear much lower intensities of the sound used than at the beginning of their investigation. They attributed this to increased familiarity with the sound and with the conditions of the test.[3]

The one certain conclusion is that hearing which is not under ordinary conditions adequate for the natural development of speech may, through special methods of teaching and training, be made of vital importance to that end.

The following records were obtained by testing patients Nos. 1 and 2 with the audiometer ('Tunnel Test'), at intervals from July 1928 to March 1929. An interesting but unfortunate feature in each case was the temporary loss of acuity gained, as the result of an attack of influenza.

[1] e.g. the Zünd-Burguet method. See bibliography 'Cathcart'.

[2] *Volta Review*, xxix. 617–18.

[3] Smith and Bartlett, 128.

TABLE G. IMPROVEMENT IN AUDITORY ACUITY OF HIGH-FREQUENCY DEAF CHILDREN DURING TEACHING BY SPECIAL METHODS

No. 1.

Frequencies:	64	128	256	512	1,024	2,048	4,096	8,192
1928, July 16th.	20	35	35	60	65	65	nil.	nil.
	25	45	40	65	70	75	,,	,,
Sept. 10th.	20	15	35	70	65	70	,,	,,
	20	15	35	65	70	80	,,	,,
Dec. 18th.	15	10	20	25	50	65	?	,,
	20	15	45	55	50	75	?	,,
1929, Feb. 21st.	20	20	45	55	50	70	?	,,
	25	15	35	40	70	65	?	,,

(The patient had been in bed with influenza in the previous week. A further cold developed before the next test.)

1929, Mar. 1st.	25	15	45	65	65	70	?	nil.
	25	20	45	75	60	80	?	,,
,, 14th.	20	15	40	35	70	70	?	,,
	15	10	45	60	55	70	?	,,
,, 25th.	10	10	45	55	60	70	?	,,
	20	15	50	65	55	75	?	,,

The readings for 4096 v.d. marked with ? indicate that there were responses at this frequency to 80, the maximum intensity of the audiometer, but that on repetition there was no reaction.

No. 2.

1928, July 16th.	35	35	35	55	90	70	nil.	nil.
	25	35	25	45	85	nil.	,,	,,
,, 26th.	30	35	35	55	85	80	,,	,,
	25	30	35	50	85	nil.	,,	,,
Sept. 10th.	30	35	30	55	90	80	,,	,,
	30	25	30	50	85	nil.	,,	,,
Dec. 18th.	20	20	40	50	85	75	,,	,,
	20	20	20	55	90	75	,,	,,

(The next test was made after the patient had been kept at home for a fortnight by an attack of influenza).

Frequencies:	64	128	256	512	1,024	2,048	4,096	8,192
1929, Mar. 2nd.	25	30	35	55	90	80	nil.	nil.
	25	20	30	60	90	nil.	,,	,,
,, 9th.	30	30	35	55	95	85	,,	,,
	25	25	25	60	90	nil.	,,	,,
,, 14th.	30	25	30	55	95	80	,,	,,
	25	25	30	60	95	nil.	,,	,,

In evaluating this table, allowance must be made for changing conditions in the individual and in the environment which cannot be controlled. The single steps in intensity represent slight differences. Fluctuations in health, especially the incidence of colds, react in a marked degree on the whole behaviour of these patients. Even with the telephone receiver as the ultimate source of sound, weather changes probably influence the results. The different factors probably affect the records in different ways. Nevertheless Table G presents certain constant features. The readings for both patients have definitely and markedly improved for the laryngeal tone 128 v.d. This improvement has remained in spite of the influenza attack with its after effects. Perhaps it is not a coincidence that 128 v.d. is the frequency on our audiometer representing the fundamental range of my voice to which they attend more often and most carefully in the course of special teaching. There is also a definite improvement at 64 v.d. With No. 1 there is a tendency to gain on other frequencies. It was pronounced at the end of 1928, and there was some sign of its reappearing in March 1929.

Further evidence in support of the suggestion that positive improvement in auditory acuity may be brought about by suitable training is afforded by the results of an experiment made with patients Nos. 227, 202, 222, 223, 242, 244, 263, and 245 (Table C, pp. 42–4), by Mrs. Ewing and Dr. H. C. Smith. The results are at present unpublished. These children were given daily 'listening-practice' from December 1928 to February 1929. The result was an improvement in acuity, in almost all the readings, of one to two steps at the end as compared with the beginning of the period.

The reactions obtained to sound were most striking, even in the short period of 2–3 months. No. 244, before the experiment, did not know that she could hear. No. 245 thought she could hear a whistle but no other sound. Yet they learned as the result of the special teaching to sing five notes up and down the musical scale, to recognize vowels by ear alone (but OO and EE were often confused, see pp. 56–64). Corresponding results were obtained with the other children. No. 263 learned to distinguish all the consonants by ear except F and TH.

The present research, therefore, taken in conjunction with

the other experiments mentioned, indicates the possibility that not only children who are high-frequency deaf, but also children suffering from previously known forms of deafness, can be given the only form of stimulus—the experience of speech heard—which leads to natural development of the speech-language function, by raising the intensity of the sounds used above the level of their thresholds at different frequencies.

Inevitable problems arise as to suitable apparatus and methods, inertia and fatigue under pathological conditions.

A summary of the conclusions drawn in Part I will be found at the end of the book.

PART TWO

INVESTIGATION OF APHASIC SYMPTOMS

CHAPTER V

THE LITERATURE OF APHASIA

INTRODUCTORY

In the first Chapter of this book an outline was given of the nature of the problem. This was shown to involve two questions:

(i) were these children deaf?

(ii) were these children aphasic?

Part I describes the means adopted to find an answer to the first question, by a series of experiments introducing new methods and apparatus.

The results showed a clear distinction between the 'high-frequency deaf' (Nos. 1–6) and other patients (Nos. 7–10) who before being examined by the new tests appeared to be suffering from similar defects of speech and language. The former showed a marked lack of hearing for sounds with higher frequency than 256 v.d., though their acuity was normal or relatively normal for notes of lower pitch. The latter proved to have perfectly normal hearing capacity. Data as to the physical analysis of speech, a number of original experiments carried out with some 110 individuals in addition to the ten above-mentioned, and a careful examination of the results of tests of hearing capacity by other workers, gave apparently conclusive evidence that the speech-language defects (pp. 1–3, 63–6, 115–18) of the high-frequency deaf children were due to their unusual condition as regards auditory acuity.

Dr. H. C. Smith's report on the condition of the ears of these patients, taken in conjunction with the fact that audiometer tests by Bunch and by Nemzek and Hays led them to an unhesitating conclusion that loss of hearing for higher but not for lower notes is associated with certain forms of inner ear and auditory nerve trouble, was shown to raise a further problem. At least three of the patients with no history of serious illness or injury were found to have no symptoms of disease of the ear. There was no reliable means of determining whether the defect was cochlear, at some level of the

auditory path, or cortical. Was it possible, in this research, to gain further information as to the aetiology of high-frequency deafness?

With the high-frequency deaf children, we had definite symptoms of lack of hearing, expressible in quantitative terms, associated with characteristic behaviour, which to a large extent was shown in Part I to be also capable of expression in quantitative terms, and on the other hand a doubt as to the anatomical seat of the trouble.

As to the aetiology of the defects of the patients Nos. 7–10 there was only, up to this point, negative evidence—they were not deaf. It should be mentioned that these four patients had been selected from a number who had become available from time to time, as presenting clearer pictures than most. In the absence of a history of illness or injury it seemed likely that there was a defect of central origin. Medical examination had led to the discovery of no deficiency either of hearing, vision, or in the organs of speech. Health had been good. Family history, so far as it was known, afforded no explanation.

The condition of these four patients (Nos. 7–10), therefore, was at this stage described as aphasic.

A careful analysis of the behaviour as to speech and language of these four 'aphasic patients', on the one hand, and of the high-frequency deaf children (as they had learnt to use speech and language in response to special teaching) on the other, was the next step.

First of all I wished to find out whether the speech-language behaviour of the two groups was really similar or whether it only appeared superficially so before analysis. In this connexion it must be remembered that Nos. 1 and 2 before these tests were described as possibly, Nos. 3 and 4 as definitely, aphasic. Secondly, therefore, it was necessary to consider the defects of the high-frequency deaf and other children in the light of the literature of aphasia and of data obtained from adult aphasic patients.

Now, if the speech-language behaviour of either group of children should prove definitely unlike that of patients described by previous workers on aphasia it might be possible to find distinctive features in their behaviour which would

powerfully supplement the tests with the audiometer in future schemes of diagnosis and enable the avoidance of such confusing and contradictory diagnoses as those referred to in this Chapter and in Chapter I.

If, on the other hand, points of similarity should be found, it would be necessary to ask whether the lack of hearing of the high-frequency deaf children was a symptom of a central defect, complicated by other features in some of the cases, but better described as aphasia than deafness, and whether the non-deaf patients were true aphasics.

The literature of aphasia is extensive, and to give a logical analysis of the conclusions of the principal investigators, within reasonable limits of space, is extremely difficult. It is unfortunate that the term aphasia has come to be applied both to defects in speech found in adults previously 'normal', but affected by an ascertained cerebral lesion, and to deficiencies in speech and language displayed by children in whom there is no direct evidence of disease, injury, or lack of physical development. In cases of 'congenital aphasia', noted almost entirely among children, the physical nature of the trouble has been conjectured on the basis of the effect on speech or language occurring in adults. Yet there appears to have been no systematic attempt to analyse and compare the behaviour of the two classes of patients. A common ground has been rather presumed than proved.

In Chapter VI an attempt has been made in this direction.

The literature of aphasia in adults, due to cortical lesions, has been taken first on historical grounds.

(1) BROCA

On April 18th, 1861, Broca, the distinguished French anatomist, made a communication to the Société Anatomique which was to initiate a controversy which is still active to-day. He said: 'There are cases where the general faculty of language persists unaltered, where the auditory apparatus is intact, where all the muscles, without excepting those of the voice and of articulation, obey the will, and where nevertheless a cerebral lesion abolishes articulate language; this abolition of speech, in individuals who are neither paralysed nor idiot,

constitutes a symptom so distinctive that it appears to me useful to describe it under a special name. I will give it the name of aphemia ('a' privative, 'phemi'—I talk, I pronounce), for what is lacking in these patients is solely the faculty of articulating words. They hear and understand all that is said to them. . . .'[1]

The cerebral lesion he localized definitely 'in the most anterior part of the frontal lobe, not far from the eyebrow and above the orbital arch', 'in the second or third frontal convolution, probably the latter'.[2]

In the language of modern science, Broca believed that he had discovered a constant correlation between characteristic symptoms of disorder in the living patient and a precisely localized cerebral lesion visible at autopsy.

METHODS OF INVESTIGATION

The majority of subsequent investigators in this field have pursued the same goal. The problem naturally presents itself under three heads:

A. Analysis of the phenomena through which the disorder is manifested in the behaviour of the living patient. This is primarily a psychological problem, and it will be shown that the more psychological the methods and standpoint of the worker the more fruitful have been his results under this heading. In recent times invaluable data have become available through the study of speech development and processes in the normal individual.[3] As yet the necessity of utilizing these data in research on aphasia has only been realized by a few workers.

B. Localization of the site and extent of the lesion responsible for the disorder. This initially recognized as an anatomical problem has more recently[4] been shown to involve physiological principles. Complex factors have also been introduced through data obtained by experimental and microscopic methods. It is improbable, for instance, that a lesion will ever be found confined to the Betz cells.

C. Comparison of the results of A and B. The correlation

[1] Moutier, 19. [2] Ibid. 19–20. [3] See pp. 4–10.
[4] See pp. 93–6 below.

aimed at here might be described as one of the supreme correlations—the elucidation of the relationship between speech, the human characteristic, and its infinitely delicate and complex mechanism in the central nervous system.

It will be noted that aphemia was the name given by Broca to the disorder he described. Trousseau, a contemporary, was persuaded that 'aphemia' signified 'infamy' in Greek, and introduced the classical 'aphasia' (=speechlessness, see Liddell and Scott's *Lexicon*) as a substitute.

In spite of violent protests by Broca 'aphasia' has survived 'aphemia' in general use.

(*a*) *Broca's methods and results of clinical examination.*

Eventually Broca and his friends examined a total of 20 patients,[1] but his deductions were mainly founded on observations of two patients, Leborgne ('Tan') and Lelong.[2]

The patients were examined for traces of paralysis, especially of the tongue, cheeks, larynx, and pharynx. Hearing acuity was tested by the tick of a watch; vision was examined.

Understanding of words was judged chiefly by the patients' attempts to answer questions. Leborgne and Lelong replied chiefly by gesture. Leborgne was asked 'How many days have you been ill?' He replied, by opening and closing his fingers, sometimes 'five' days and sometimes 'six' days. 'How many years have you been at Bicêtre? He opened his hand four times in succession and then raised one finger, this made 21 years, an answer which was perfectly correct.' He replied thus to the same question on two successive days, but when it was repeated on the third day 'he became angry and articulated (an) oath'.

Speech.[3] Questions were asked. Leborgne was nicknamed 'Tan', because that word was, with rare exceptions, such as the oath just mentioned, the only word he could use. Lelong said 'Oui' and 'Non', 'tois' (trois) and 'toujours'. These two patients suffered from severe and widespread disorder. It will be shown that though later workers have revolutionized Broca's methods of psychological examination his data were of value.

[1] Head (3), vol. i. 26.

[2] Moutier, 17–25, and Head (3), vol. i, chap. 2, contain considerable extracts, from Broca's own papers.

[3] Moutier, 17–25.

(*b*) *Site and extent of cerebral lesions.*

Leborgne: a cavity corresponding to the fissure of Sylvius extended back to, but was entirely in front of, the fissure of Sylvius. The capacity was equal in volume to that of a hen's egg.[1]

Lelong. 'Left frontal lobe, area equal to that of a franc.' The 3rd frontal convolution was cut in half. The 2nd frontal convolution was profoundly eaten away.[2]

These lesions were of a most extensive nature. About 1906 Marie re-examined the two brains and came to different conclusions as to the topography of the most serious damage (p. 92 below).

(*c*) *Conclusions.*

Broca compared the symptoms, which he concluded to be loss of 'the faculty of articulating words' without loss of understanding or of the use of the speech organs for functions other than speech, with the cerebral lesion which (in 1861) he located 'in the most anterior part of the frontal lobe'. He adopted the 'faculty' psychology, then widely current, and conceived speech as having a narrowly limited and localized representation in the cortex. But his hypothesis cannot be assessed at its true importance without remembering that the medieval doctrine of three ventricles in the brain, each the dwelling-place of an aspect of the soul,[3] was still an influence in early nineteenth-century medical teaching. Lelut and others hotly opposed the very conception of cerebral localization of function.[4]

Broca's debt to the often despised Gall (1758–1828) has been traced by Head—'we are really indebted (to Gall) for the ideas we now hold of the relation of the constituent parts of the nervous system to one another.'[5]

Before the end of 1864 Broca attended two post-mortems of patients who had exhibited symptoms of what he described as 'aphemia'. He was astounded, however, to find that in the one instance the lesion occupied the neighbourhood of the posterior portion of the fissure of Sylvius, 'and that in the other there were several lesions, . . . none of which affected the

[1] Head (3), vol. i. 17–25. [2] Ibid. [3] Head (3), i. 1.
[4] Moutier, 26–32. [5] Head (3), i. 4.

3rd frontal convolution.'[1] He pointed out, nevertheless, that among the 20 cases collected by himself and his friends all showed some pathological change in the left half of the brain.[2] This was a recognition of what has since come to be known as the theory of 'the dominant hemisphere' (pp. 87–8 and 108–9 below). Neither Broca nor his friends appear to have known that Marc Dax had read a paper on the subject at Montpellier in 1836.[3]

Broca attempted to interpret the evidence of these two post-mortems by distinguishing between aphemia, a disorder caused by lesion of the 3rd frontal convolution, and 'verbal amnesia', in which the patient could pronounce words but had lost the special memory of spoken and written words.[4]

This modification of his original thesis was put forward at the British Association Meeting at Norwich in 1868. He was followed at that meeting by Hughlings Jackson.

(2) HUGHLINGS JACKSON (published work 1864–93)[5]

Jackson approached the study of aphasia from a strictly phenomenal standpoint. He was a psychologist as well as a physician and pathologist, yet he was careful not to confuse physical and psychological phenomena; 'we must not classify on a mixed method of anatomy, physiology, and psychology;'[6] 'we must be on our guard against the fallacy that what are physical states in lower centres fine away into psychical states in higher centres; that . . . vibrations of sensory nerves become sensations;' but we must endeavour 'to trace a correspondence'.[7]

Head has remarked: 'Even the most modern accounts, such as that in von Monakow's Monograph (see pp. 93–6 below), attempt to deal with the clinical facts as if they could be deduced from the physiological activity of morphological centres.'[8]

Jackson's psychology was drawn in the first place from Herbert Spencer,[9] but his hypotheses were the product of personal observation. Yet 'he never experimented or arranged a series of observations to elucidate a definite point'.

[1] Head (3), vol. i. 26. [2] Ibid. i. 26. [3] Ibid. i. 28.
[4] Ibid. i. 27. [5] Jackson, see Bibliography. [6] Ibid. 115.
[7] Ibid. 109. [8] Ibid. Introd. 4. [9] Ibid. 44.

'He would watch a patient in the throes of delirium tremens as he tossed in the padded room, or listen to . . . the recurrent utterance of some partly demented aphasic.'[1]

A. *Results of clinical observations.*

'A patient may do a thing well at one time and not at another,'[2] e.g. 'a few months ago a patient came under my care who could only say "pooh! pooh!" and an examination of his eyes was almost impracticable. . . . Instead of opening (his eyes), he opened his mouth, . . . although he . . . was all the time doing something with his muscles.'[3]

Jackson recognized, therefore, the difficulty of assessing the general understanding of some patients. 'Now it may be said that the patients do not know what we wish them to do . . . but . . . a speechless patient who cannot put out his tongue when told will sometimes actually put his finger in his mouth as if to help to get it out.'[4] Yet 'In the ordinary "specimen" of loss of speech . . . the patient cannot speak at all, but understands all we say, on simple matters at any rate.'[5]

Jackson distinguished between what he called 'higher' and 'lower' speech. Herbert Spencer had written 'All speech is compounded of two elements, the words and the tone in which they are uttered, . . . certain articulations express the thought, certain vocal sounds express the more or less of pain or pleasure which the thought gives.'[6] Jackson found that the defect described as 'aphonia' involved loss of voice with ability to whisper speech retained and was never due to a lesion of the hemisphere.[7] On the other hand, he noted a patient whose speech was unintelligible jargon and yet the intonation was so varied that it clearly expressed her natural emotions.'[8] He was in contact here with a pathological problem as yet almost unexplained (cf. p. 57). But he passed on to a different though related aspect. He noted the ability of aphasic patients to swear and sing. He was able to quote examples of 'speechless' patients who spoke under the influence of strong emotion. He concluded that swearing, interjections, and 'emotional' speech formed a lower 'auto-

[1] Jackson, 6.
[2] Ibid. 48.
[3] Ibid. 49.
[4] Ibid. 50.
[5] Ibid. 78; cf. 120.
[6] Ibid. 44.
[7] Ibid. 33.
[8] Ibid. 45.

matic' type of utterance.[1] 'Propositionizing' is a higher form. He recognized two forms of propositionizing, in words —'a proposition is such a relation of words that it makes one new meaning'[2]—and in images.

At this distance of time, with great changes in psychological teaching intervening, it is difficult to understand Jackson's use of the word 'proposition'. But these facts seem to be clear, first that as a rule he was thinking chiefly of the capacity for putting thought into words, without necessarily giving them utterance—'the speechless man' cannot write 'because he has no propositions to write', though 'in most cases he can copy writing', and similarly because he cannot apprehend propositions 'he cannot . . . in most cases . . . read at all'.[3] In such cases 'perception (or "recognition" or "thinking" of things) (propositions of images) is unaffected, at any rate as regards simple matters', for the patient plays cards or dominoes and 'he recognizes handwriting though he cannot read the words written'.[4]

Secondly, Jackson seems to have considered synthesis an essential factor in the act of making or apprehending a proposition, 'when we apprehend a proposition a relation between two things is given to us'.[5]

These two features of his teaching alone show his awareness of the complexity of the normal thought processes and of the disorders due to aphasia. He stood remote from the theories of a 'faculty' of speech based on a strictly localized 'centre' in the brain and the accompanying conception of the unexpressed use of words as a strictly intellectual phenomenon.[6] To-day we may reject 'images' as essential to thought and we may hold that consciousness plays a lesser part in controlling behaviour, but we must grant that Jackson touched the root of the matter when he realized that an aphasic patient might think clearly without words and that his trouble is essentially a breakdown in his capacity for synthesis (see p. 103 and p. 143 below).

Jackson distinguished three degrees of severity in the disorders known as aphasia.

(1) 'Defects of speech'. The patient who 'has a full

[1] Jackson, 51, 52, 77, and 78. [2] Ibid. 113. [3] Ibid. 119.
[4] Ibid. 121. [5] Ibid. 66. [6] Head (3), i. 28.

vocabulary, but makes mistakes in words, as saying "orange" for "onion", "chair" for "table", or uses approximative or quasi-metaphorical expressions, as "Light the fire up there" for "Light the gas".'[1]

(2) 'Loss of speech'—the 'speechless man' already described who can often neither read nor write.

(3) 'Loss of language'—the patient 'has altogether lost pantomime, and emotional language is deeply involved'.[2]

'There can be no basis for comparison betwixt the wrong utterances in No. 1 and the non-utterances in Nos. 2 and 3. . . . There is a negative and a positive condition in each degree. . . . The condition of the patient No. 1 . . . was duplex, (*a*) negatively in not saying "table", and (*b*) positively in saying "chair" instead; there is . . . loss of some speech . . . with retention of the rest of speech . . . it is the best speech under the circumstances and is owing to the activity of healthy (except perhaps slightly unstable) nervous elements.'[3]

There is 'dissolution'[4] and 'the presumption is that the patient uses what is to him a more organized or "earlier" word'.[5] Jargon Jackson described as the 'survival of the fittest' syllables.[6]

This remarkably 'modern' analysis fails to cover one possibility—the patient who having called 'table' 'chair' (as above), a few minutes later reverses the performance (see one of my own patients, p. 137 below).

'Pantomime' Jackson included as a branch of language and discussed its origin in sensory sensation.[7]

In addition he described 'a morbid condition in the image series (imperception) which corresponds to aphasia in the word series'—'in simple cases of delirium . . . when a patient takes his nurse to be his wife' he is reduced to 'lower, earlier and prior stages, of perceiving and remembering.[8] He refers to 'word-deafness (a special imperception)'.[9]

B. *Site and extent of cerebral lesions.*

In his first paper in 1864 Jackson brought forward 34 cases, but he had not had a single post-mortem.[10] Nine years later

[1] and [2] Jackson, 116. [3] Ibid. 117. [4] and [5] Ibid. 111.
[6] Ibid. 162. [7] Ibid. 180–1. [8] Ibid. 125.
[9] Ibid. 176. [10] Ibid. 28, 29.

he wrote in the *Lancet* (June 18th, 1873, p. 84) 'On the anatomical and physiological localization of movements in the brain', in which he refers to 'studies of cases of disease of the brain . . . for anatomical and physiological purposes'.[1]

Before Broca's first communication Jackson had concluded from instances of the loss of speech attending a condition of right hemiplegia that the activity of the left hemisphere is essential to 'voluntary' speech. He found this confirmed by the occurrence of left hemiplegia with loss of speech in left-handed patients,[2] but admitted that there are exceptional cases; 'loss of speech has occurred from damage . . . on the right, and the region on the left has been damaged without any affection of speech'.[3]

The left third frontal convolution is the part most often damaged, but the lesion 'which destroys speech is always in the region of the corpus striatum' and is usually damage to but one hemisphere.[4]

C. *Conclusions on comparing A and B.*

In opposition to the prevailing contemporary conception, which at its crudest appears to have amounted to the idea that so much cerebral substance lost meant so many words lost, Jackson wrote: 'I do not believe that words or syllables have nervous arrangements in the sense that there is one little nervous centre for each syllable, or rather single articulation. . . . But a certain quantity of nervous arrangements . . . is required for each operation.'[5]

Nevertheless he correlated recurring utterances in otherwise speechless patients, the occurrence of emotional speech (see p. 84) and the facts of hemiplegia just mentioned, and deduced that 'it is not possible that such a (recurring) utterance was effected during discharges . . . of any part of a half of the brain which was so much damaged as to cause speechlessness'.[6]

He said that when the patient is speechless, or nearly so, disease tends to lie in the neighbourhood of the corpus striatum, but that if words are plentiful and yet there are mistakes in the words, the disease reaches further back or deeper in the brain.[7]

[1] Jackson, 75. [2] Ibid. 63. [3] Ibid. 81. [4] Ibid. 81.
[5] Ibid. 155. [6] Ibid. 156. [7] Ibid. 24.

Disease of the left corpus striatum therefore 'permits the increased dischargeability of the right half', and lower automatic speech results from this removal of inhibition.[1] The right half is also involved in the reception of words.[2]

Jackson therefore was in agreement with Broca as to the regions of the brain most often affected by lesions productive of aphasia (p. 82). But his extensive clinical studies led him to a much wider comprehension of the complexity of the psychological, physiological, and anatomical structure involved. He was led by his observations to realize, as Broca could not (p. 83), that lesions neither in the '3rd frontal convolution' nor in the neighbourhood of the corpus striatum might affect, even if they could not 'destroy', speech.

3. BASTIAN[3]

Bastian was the chief representative in England of a school of thought which exerted great influence on the teachings of neurology.

He definitely stated his opposition to the conclusions of Hughlings Jackson.[4] But the methods and standpoint of the two workers were so completely different that comparison is difficult.

In 'central nerve units' a 'particular retentum is stored up or registered'.[5] 'Retentum' was a term coined by Hamilton to describe purely psychological processes associated with remembering.[6] This identification of psychological with physiological and anatomical phenomena led to the dogma of 'central nerve unities', each corresponding to what Bastian and his followers identified as constituent psychological elements in the production of speech and language.

'A number of underlying problems will have to be studied before the different kinds of speech defect can be individually studied',[7] he writes in his first chapter. He makes a number of general statements about the development of speech in the normal child, e.g. 'the great majority of children can remember the names given to many external objects when they are four or five months old',[8]—this is wholly inconsistent

[1] Jackson, 158. [2] Ibid. 176. [3] See Bibliography.
[4] Bastian, 81, 93 and 270–3. [5] Ibid. 12. [6] Ibid. 10.
[7] Ibid. 2. [8] Ibid. 4.

with the findings of any well-known authority on the subject: so also was his theory that 'speech has now become a truly automatic' act in human beings and is comparable with the sucking instinct.[1]

However Bastian recognized that the capacity and experience of hearing words spoken is the stimulus productive of speech in the young child.[2] Reading and writing are superadded accomplishments.[3]

There are 'three distinct kinds or physiological types of memory . . .'[4]

(1) Auditory, the memory of the sound of words;

(2) Visual, the memory of their 'visual appearances (printed or written forms of words)';

(3) Kinaesthetic, (*a*) 'memory of different groups of sensory impressions resulting from the mere movements of vocal organs'. (*b*) 'Memory of the different groups of sensory impressions emanating from muscles, joints, and skin during the action of writing individual letters or words.'

'The organic seat of each of these four different kinds of word-memory is in relation with its own set of afferent fibres.'[5] This was the origin of the famous diagram. Other members of the same school of thought produced similar, often much more complicated diagrams on the same basis.

Since Bastian's day a period rich in experimental, clinical and comparative studies in neurology has intervened, making a detailed discussion of his works unnecessary. It is clear that his standpoint was neither phenomenal nor genetic. The diagram was not based on clinical observation but on the arbitrary division of the speech-language function into hearing, seeing and feeling, speaking and writing words. This arbitrary division was then attributed to the nervous system, and it was concluded that each of these hypothetical functions was represented in a distinct region of the cortex. There seems no doubt that in diagnosis Bastian asked himself 'Is the patient's auditory word-centre, visual word-centre, glosso-kinaesthetic, or cheiro-kinaesthetic word-centre, or the connexions between any two of these word-centres affected or destroyed?' (See his *Clinical Characteristics*, pp. 311–14.)

[1] Bastian, 6. [2] Ibid. 3. [3] Ibid. 3.
[4] Ibid. 12. [5] Ibid. 13.

Only a small percentage of his 113 records of patients were based on personal observation. An analysis of the records (e.g. No. 58[1]) suggests that Bastian had difficulty in fitting his patients into his ready-made scheme of nine categories of defects of speech associated with cerebral lesions.

Similarly Head relates[2] how Bastian had at University College Hospital from 1878–96 a patient (No. 94[3]) whom he was in the habit of demonstrating to students, explaining with the help of the diagram exactly what 'commissural' (i.e. connecting 'word-centres') fibres were affected. However, as the result of the post-mortem Bastian himself wrote: 'The difficulties in reconciling the persistent and often verified clinical condition with the post-mortem record (are) extreme.'[4] The extent of the lesion was far greater than he had anticipated.

4. MARIE AND MOUTIER

It was unfortunate that the diagram-making school were inspired by a truly 'arm-chair psychology', especially since they dominated teaching on aphasia for some forty years.

But in 1906 Pierre Marie attacked not only the dogmas of the 'centres and commissures' school, but even the original conclusions of Broca himself—'The third convolution does not play any special role in the function of language',[5] was the title of a paper by him in the *Semaine Médicale*.

Two years later Moutier published his monograph, 'L'Aphasie de Broca',[6] a comprehensive and authoritative summary of his master Marie's teaching.

A. *Methods and results of clinical examination.*

A systematic and comprehensive scheme for examining patients was included.[7]

Hearing was tested with the watch and whispered speech.[8] Every examination must be repeated, but each repetition must be brief.[9]

Moutier gave the results of 44 'personal observations' of patients made on these lines and also collected records of 387 patients made by other workers.

[1] Bastian, 176.
[2] Head (3), i. 57.
[3] Bastian, 255–60.
[4] Ibid. 260.
[5] Head (3), i. 67.
[6] See Bibliography.
[7] Moutier, 375–83.
[8] Ibid. 376.
[9] Ibid. 206.

His conclusions were:

(1) The aphasic patient whose speech is affected is free from paralysis but suffers from symptoms of incoordination of the speech organs.[1]

(2) The extent to which articulate language is 'abolished' varies. The patient may be incapable of a cry, his speech may have a strongly emotional character, he may jargonize, use isolated words, or repeat the same words again and again. These were Jackson's conclusions also (pp. 85–6 above). Under the influence of an emotional shock the patient may use words which he cannot repeat afterwards.[2] There are certain patients who can complete words when the first syllable is suggested to them. Aphasics can sing an air, but not always the words of a song.[3]

Reading and writing are as a rule affected.[4] Again Moutier agreed with Jackson. Powers of calculation are considerably impaired.[5] Some mute patients can express themselves marvellously in gesture, but this power may be affected (see Jackson also, p. 86 above).

(3) There are patients not paralysed, whose speech is affected, but whose understanding is perfect. They suffer from a motor incoordination. These are not cases of aphasia proper, but of anarthria. The aphasia of Broca is a syndrome, a combination of anarthria and aphasia.[6] Eight instances of 'anarthrie pure' are given among Moutier's 'personal observations'. Of these No. XIII[7] 'was absolutely speechless but his mental reading was perfect'. This is the most convincing instance, though there are no details about the 'mental reading'. The clinical picture of 'anarthrie pure' in the other seven cases is not distinct. For example, No. VIII[8] was an almost illiterate man of 75 years, who 'executed correctly orders given him to carry out, and if he happened now and then to commit some error in detail, it was that his good will had been surprised in some manner and he at once corrected his first mistakes'—which is not convincing proof that he had completely understood the command.

(4) This distinction between 'anarthrie pure' and 'aphasie' suggests a physiological—motor and sensory—basis (see

[1] Moutier, 191. [2] Ibid. 194. [3] Ibid. 197. [4] Ibid. 197.
[5] Ibid. 199. [6] Ibid. 249. [7] Ibid. 551. [8] Ibid. 466–8.

Moutier's localization teaching below). But Moutier insisted that 'Aphasia is a disturbance of interior language, and of all its modes of exteriorization, it affects the word, the intellectual symbol'.[1] Of 'images' he said 'the image buries itself in the complexities of thought'. Aphasia is 'a general intellectual defect and a special intellectual defect for language'.[2] Yet it is emphatically not a form of dementia.[3] The complexity of the problem with which the aphasic is faced is an important factor in determining the efficiency of his reaction, whether in fulfilling a command, in understanding what he reads, or in repeating words.[4] 'The aphasic is in the situation of a man in a strange country speaking the native language with difficulty. Shall we say that this man is affected with word-deafness because he cannot understand some one speaking to him too quickly, using too long phrases, employing too numerous words or phrases? Clearly not.'[5]

Thus Moutier's analysis of his clinical data agreed with Jackson's to a remarkable extent.

B. *Site and extent of the lesions.*

Marie, on re-examining the brains of Broca's patients 'Tan' and Lelong, found that in the first the lesion was most extensive and included the lenticular zone (corpus striatum and insula), the greater part of the first temporal, and to a considerable extent the supra-marginal gyrus.[6] In the second there was 'a lesion, abnormal in its very nature, of the foot of the two frontal convolutions, inferior and middle, side by side with an equally indefinite lesion of the left corpus striatum'.[7] In 24 'personal observations with autopsy' and either macroscopic or microscopic examination of the brain[8] there was a large proportion of very extensive lesions. Those described were in the '3rd frontal convolution', 'lenticular zone', and supra-marginal gyrus.

C. *Conclusions on correlating.*

Moutier laid stress on the existence of lesions of the 3rd frontal convolution of the left hemisphere in right-handed

[1] Moutier, 205. [2] Ibid. 220. [3] Ibid. 228. [4] Ibid. 221.
[5] Ibid. 211. [6] Ibid. 76. [7] Ibid. 77. [8] Ibid. 384–599.

patients without defects of speech or language.[1] But among his own three instances the evidence of normal speech was not detailed. He claimed that among 108 cases in medical literature 84 contradicted the localization of a 'speech centre' in the frontal lobe.[2] 'The defect of articulate speech, anarthria, depends on a lesion of the lenticular zone' (roughly the corpus striatum and insula). The theories of 'gaucherie cérébrale' (i.e. a 'speech centre' in the left hemisphere of right-handed individuals), of substitution and of re-education, contain a considerable amount of hypothesis.[3] The aphasia of Broca is the aphasia of Wernicke+anarthria. The aphasia of Wernicke was held to be associated with a lesion of the zone of Wernicke,[4] i.e. in the temporo-parietal region.[5]

5. HENSCHEN (publications 1918, 1919, 1922, &c.)

Henschen successfully correlated limited lesions in the calcarine region with quadrantic defects of vision; and his conclusions were confirmed by Gordon Holmes and Lister during the war period.[6] Subsequently Henschen made an investigation of pathological cases and determined that the 'primary centre' for hearing was in the gyrus transversus and not in the 1st or 2nd temporal convolution as had previously been thought.[7]

6. VON MONAKOW (publications 1909, 1914, 1922, &c.)

'The key-note of his position is given by the universally recognized fact that the local symptoms which immediately follow a non-progressive lesion are severer, more extensive, and often less sharply determined than is the case after the expiration of some days or weeks'[8]—'a cortical lesion can be followed by defects of function which are of sub-cortical, or even of spinal, origin.'[9] From this basis and, after long investigation, Von Monakow developed the doctrine of diaschisis. The essence of this is that the symptoms caused by local injury are due to heightened resistance at the synapses or nerve junctions. The following translation is due to Head.[10]

[1] Moutier, 245. [2] Ibid. 245. [3] Ibid. 247. [4] Ibid. 249.
[5] Ibid. 142. [6] See Bibliography. [7] Head (3), i. 78.
[8] Ibid. i. 85. [9] Ibid. i. 85. [10] Ibid. i. 86.

'Diaschisis consists of a suspension of activity, which usually arises suddenly, affecting a widely radiating central field of function. It takes its origin in the local lesion, but unlike apoplectic shock does not attack the whole cortex; its incidence falls on those parts only where fibres from the site of the lesion terminate in primarily uninjured grey matter of the whole central nervous system. Diaschisis consists essentially of a lowering or abolition of the power of the central elements (groups of neurones) to respond to stimuli within a definite and physiologically definable zone of excitation; this zone, however, does not correspond with the usual physiological paths of innervation which extend either from the periphery or from the centre.'

This factor therefore must be taken into consideration in studying records of clinical observations of aphasic patients made within some weeks or months of the incidence of the lesion.

In regard to cerebral localization, Von Monakow concluded, firstly, that of various movements or qualities of sensation, 'those which are lower in form, more generalized and acquired earlier in the life of the individual are the first to be restored'.[1]

Secondly, that the only form of anatomical localization which can be unhesitatingly accepted is the relative topographical representation of the various parts of the body.[2] Thirdly, 'When we attempt to deal with a succession of synergic movements . . . the complete act in its perfect form demands the mobilization in due sequence of a series of complex procedures; . . . the time relation . . . is of fundamental importance.'[3] '. . . Among the complex excitations which evoke acts of speech, occurs a new factor, the symbol; this is a shortened sign for the widest possible combination of stimuli, different in aim, origin and feeling.' 'Speech is developed step by step in the life of every individual and a temporal character is impressed on it from the beginning.'[4]

Thus Von Monakow substituted the conception of a highly complex system with strata upon strata of development for the theory of 'word-centres' or the 'speech-centre'.

Clinical localization, however, he found possible. When 'after many weeks or months' diaschisis has passed away

[1] Head (3), i. 87. [2 3] Ibid. i. 88. [4] Ibid. i. 89.

'residual symptoms . . . permanent disintegration, disturbances of rhythm and lowered excitability remain'.[1]

There are two groups of symptoms:

'Motor' aphasia: the patient 'can swear and give vent to expressions which have become automatic . . . may be able to write his name or single letters and portions of words'.[2] But though he can follow ordinary conversation, he cannot apprehend the significance of a logical sequence; this applies in a still higher degree to written or printed matter. 'But, especially in cases of local injury, these symptoms pass away in the following stages:

First . . . comprehension of spoken or written words may return completely. Then the loss of power to evoke words ("dumbness") yields gradually; the patient learns to utter those in common use and can repeat them to order. But speech is uncertain, the voice monotonous, intonation defective, syllables are slurred and there is a tendency to perseveration. . . Then follows a period in which short sentences evoked spontaneously and words in common use can be said without difficulty. At this stage the patient may remain. . .'[3]

'Sensory' aphasia[4] . . . the patient cannot comprehend ordinary conversation or carry out oral commands; spoken words are to him incomprehensible noises . . . (but) he still possesses the power of comprehending the meaning of some isolated words and phrases, such as his name and "How are you? . . ."' 'He can talk spontaneously . . . and is at times even unduly garrulous . . . although uncertain internal verbalization constantly leads to errors. . . .' 'His sentences are constructed grammatically . . . but his defective powers of speech are betrayed by false intonation and accent, by rhythmic disorder and monotony of voice. The words are . . . confused mainly in accordance with some resemblance in sound. . . . Above all things, there is a tendency to perseverate'. . . . 'Repetition is impossible. . . .' 'Complete sensory aphasia invariably leads to some intellectual want of power to manipulate written speech. There is always agraphia combined with alexia. . . . Letters can be appreciated visually

[1] Head (3), i. 89.
[2] Ibid. i. 90.
[3] Ibid. i. 90.
[4] Ibid. i. 90 and 91.

and may even be written down correctly, although their sound value and significance are mistaken.'

'Writing . . . can be disturbed . . . by defective verbalization (word-dumbness) . . . by want of appreciation of the verbal significance of sounds, and lastly by . . . disturbance of the visual components in speech.

The 'most favourable situation' for the production of motor aphasia is 'the area between the posterior portion of F. 3 and the operculum. . . .'[1] 'Sensory aphasia can be produced anywhere within the temporal lobe. . . .'[1]

7. PICK

Pick in *Die Agrammatischen Sprachstörungen*, 1913, made an avowedly psychological study of the particular form of speech defect in which there may be jargon but of which the main features are disturbance of rhythm and grammatical structure. He accepted Jackson's views on 'Evolution and dissolution of the nervous system'.[2]

He analysed the processes on the way from thinking to articulate speech—'intuitive thought, the proposition, the grammatical schema and the explicit verbal formula . . . These are not elements . . . but stages in the development of a mode of behaviour, which can be interrupted or disturbed at any point in the process'.[3] He pointed out the defects of rhythm and intonation noticeable in jargonizing speech and 'laid stress on lack of inhibition as an important factor. . .'[4] His point of view was strictly genetic and evolutionary. In organic lesions he wrote that 'The higher intellectual aptitudes are the first to be lost in the order of their difficulty, whilst emotional forms of speech suffer later, to disappear just before mimicry, the ultimum moriens of expression.'[5]

The speech of the aphasic may be lowered to a level resembling that of a child, where relatively 'primitive methods of unformulated thinking'[6] prevail.

A lesion of the left temporal lobe can interfere with the correct use of syntax.[7]

Head gives a synopsis of Pick's article on aphasia (for Bethe's *Handbuch der Physiologie*) written just before his death

[1] Head (3), i. 92–3. [2] Ibid. i. 125. [3] Ibid. i. 125. [4] Ibid. i. 125.
[5] Ibid. i. 126. [6] Ibid. i. 126. [7] Ibid. i. 126.

in 1924.[1] In this he stated that 'expressive' acts of speech were disturbed by a lesion in the frontal region, impressive by 'structural changes in the temporal area'. But all 'localization of the various clinical manifestations must be of the widest general character'.

Expressive defects of language include 'gross loss of articulatory speech', inability to repeat what is said, and occasionally to write spontaneously, and interference with the capacity for turning thought into words.

'Impressive' aphasia comprises interference with the power to understand words, and consequently disturbances also of speech, reading, and writing.

There are various syndromes of disordered speech, e.g. two forms of agrammatism. A temporal variety is characterized by defective syntax, false verbal inflexions, wrong prefixes and suffixes, hurried speech. 'In that of frontal origin articulation is slow, the pauses are lengthened and the words are juxtaposed as in a telegram.'

8. HEAD

Head in 1926 published the first comprehensive survey [2] in English of the whole subject of aphasia. His monumental work included a detailed history of the work of all the principal European investigations into aphasia, a full account of his scheme of original tests and methods of investigation, an analysis of the results obtained by testing a large number of patients, a classification on an original basis of the defects discovered, a section on the localization of the lesions responsible for these defects, and another on the physiological and psychological problems involved. In a second volume he gave full clinical records of 23 patients, some of the records extending over periods of years.

Head's patients, as he pointed out in the Linacre Lecture in 1920,[3] were not 'old, broken down', and with 'general intellectual capacity diminished', like the majority of aphasic patients in civilian practice. They were war patients, 'struck down in the full pride of health' and many of them educated, extremely intelligent, and able to give important introspective evidence.

[1] Head (3) i. 126–8. [2] See bibliography. [3] Head (2).

Moreover, the vascular lesions responsible for most cases of aphasia in civil life are frequently responsible for 'profound and widespread disturbance of function', while gunshot wounds, striking the skull from without, 'give greater opportunity for the appearance of loss of function in dissociated forms'.[1]

Moreover, as 'with gunshot wounds of the head the symptoms tend to clear up to a considerable extent . . . we are enabled to trace the various steps by which the defective functions are restored, whereas in civilian practice any change . . . is usually in the opposite direction'.[2]

In arranging and giving his tests Head insisted on the necessity for experimental conditions, repetition of identical stimuli in irregular order in a series, and notes of any departure from the pre-arranged plan. Fatigue and disappointment in the patient must be avoided at all costs.[3]

The Serial Tests.

There were six 'Serial Tests'.[4] The first two (*a*) and (*b*) tested the patient's power to name and recognize common objects (knife, key, &c.) and colours. The patient was required to point to one of a number of objects or colours first named aloud, then indicated by a printed card, by the examiner. He was also asked to name aloud each object or colour as it was pointed out by the examiner, to repeat its name after the examiner, to write its name spontaneously, and to copy its name into cursive characters from print.

(*c*) The third of these tests was 'designed to investigate the power of reading and writing in its simplest and most elementary form'.[5] It was called the 'man, cat, and dog test,' and involved reading aloud, writing from dictation, 'writing from pictures', 'reading from pictures', repetition aloud, and copying 6 to 10 monosyllabic phrases such as 'The cat and the man'. In writing and reading from pictures the patient was asked to write the names or name aloud, any two of the three pictures (man, cat, and dog respectively) as presented by the observer in a certain order.

(*d*) The clock tests.[6] For these, two clock faces, marked in

[1] Head (2), 91. [2] Head (3), i. 146. [3] Ibid. i. 147.
[4] Ibid. i. 149–60. [5] Ibid. i. 153–5. [6] Ibid. i. 155–6.

Arabic numerals, with adjustable hands, were used. The patient was provided with one clock face and requested to imitate the setting of the hands on the other by the observer. He was then asked to tell the time when the observer set the hands of one of the clock faces, and to set the hands himself to oral and printed commands.

(*e*) The coin-bowl tests.[1] The patient had to place a coin in one of four bowls according to oral commands, printed commands read silently, and printed commands read aloud.

(*f*) The hand, eye, and ear tests.[2] 'First of all the patient, seated opposite the observer, attempted to imitate a series of movements which consisted in touching an eye or an ear with one or other hand.' Next the patient imitated the movements of the observer reflected in a mirror, then executed commands to make similar movements presented pictorially, then carried out these commands when the pictures were reflected in a mirror. He was also required to make the same movements in response to oral commands and printed commands read silently and aloud. Lastly, he wrote down movements made by the observer with and without the use of the mirror.

The essence of this test was the fact that Head found that normal individuals always perceived when the hand had crossed the face, but his aphasic patients made many mistakes in this respect, tending, however, to make fewer mistakes as they recovered from their wounds.

It is clear from Part II of vol. i and from vol. ii of his book that Head used these six tests as a means of standardizing his conclusions as to the forms the defects assumed in his patients. But he made great use also, especially when analysing defects of higher levels and more complex orders of thought, of supplementary tests. These were repetition, reading and writing the alphabet, days of the week, &c., counting and arithmetical tests, drawing, finding one's way about, puzzles and games, analyses of conversation, reading and writing, inquiries into appreciation of pictures and musical capacity.[3]

[1] Head (3), i. 156–7. [2] Ibid. i. 157–60.
[3] Ibid. i. 160–5 and 300–80.

Criticisms of Head's Tests.

Kinnier Wilson has objected [1] that the serial tests, especially the mirror test, are 'tests of intellectual capacity . . . rather than of speech activity in the ordinary sense'. But Head's reply [2] is that 'words are the commonest and most obvious symbols used in thinking', the aphasic patient cannot perceive crossed movement (i.e. when no words are to be spoken, read, or written) because these acts, when the patient is face to face with the observer or the picture, are propositional and involve (mental) verbalization. The patient can imitate reflections of the observer's movements or the pictorial representations of them in the mirror because 'no verbalization is necessary'—the patient has only to make movements, whether crossed or uncrossed, on the same side of the central line of the body as those he sees reflected.

This is admittedly a difficult point and affects the interpretation attached to parts of both 'hand, eye, and ear' and 'coin-bowl' tests. It is doubtful whether psychologists, in spite of the Würzburg school, can as yet solve the problem.

Head's Four Clinical Varieties of Aphasia.

The conclusions which led Head to adopt his now well-known classification of aphasia into four varieties, 'verbal, nominal, syntactical, and semantic' may perhaps prove of greater permanent value than the classification itself. For his was an attempt to study the clinical phenomena with an unprejudiced mind. Like Jackson, his standpoint was strictly phenomenal. This was not the case with Marie and Moutier, Pick, or even von Monakow, each of whom was clearly influenced by the anatomical and physiological division of 'motor' and 'sensory' functions. Head considered five of his patients in relation to this latter classification, showing that their symptoms were inconsistent with these descriptions.[3] His patient No. 20, for instance, appeared at first sight to be an example of 'motor' aphasia—'all forms of articulated speech and writing were grossly affected, but objects could be chosen correctly when the names were said by me or presented to her in print. But . . . the "receptive"

[1] Discussion on Aphasia, 434.

[2] Head (3), i. 208–9.

[3] Ibid. i. 202–10.

aspect of her use of language had not in reality escaped; for she had difficulty in setting the hands of the clock and could not carry out the hand, eye, and ear tests either to oral or printed commands.'[1]

The key to Head's position appears to be as follows: 'A man who has hurt his foot may be unable at first to stand, to walk, still less to run. As the effects of the injury pass away, he recovers . . . and can perhaps walk in some characteristically abnormal fashion. . . . But we gain nothing by stating that the patient has recovered from his "astasia" and still suffers from some "abasia" and total "adromia".'[2]

'This applies equally to the words "aphasia", "amnesia", "alexia", "agraphia", "agnosia", "apraxia" . . . they are convenient shorthand descriptions for varieties of abnormal behaviour, each of which may comprise morbid phenomena of a totally different nature and origin.' 'The various disorders of speech produced by injuries of the brain manifest the ways in which the organism masters a situation demanding the use of language with a defective mechanism.'[3]

'"Speechless" patients are not of necessity wordless. . . . A man usually speechless may at times produce a complete phrase'; 'in attempting to discover if a patient can read, we not uncommonly find that he can choose a familiar object without fail if the name is presented to him in print, although he is unable to carry out a simple command to touch some part of his body with one or other hand'; as to writing, 'sometimes a man can write his name and address correctly, but cannot give that of his mother with whom he lives'.[4]

In aphasia 'all the acts (of speaking, reading, and writing) suffer more or less, according to the severity of the loss of function.'[5]

Speaking, reading, and writing are 'purely linguistic terms for diverse human actions, and do not correspond even to specific groups of psychical functions.'[6]

Head introduced the term 'symbolic formulation and expression'[7] as being less cumbrous a description of acts of speech and language than Jackson's propositionizing.

[1] Head, i. 204. [2] Ibid. i. 301. [3] Ibid. i. 300–1.
[4] Ibid. i. 319. [5] Ibid. i. 220. [6] and [7] Ibid. i. 428.

It will be seen, therefore, that Head considered that the arbitrary and empirical divisions of the one speech-language function—understanding and uttering words, reading and writing—are so inextricably interrelated that they stand or fall together. An attempt to trace the reasons for this in racial and individual development was made in Part I of this book (pp. 11–12).

The factors affecting the 'acts of symbolic formulation and expression' of aphasic patients were said by Head to be the severity of the lesion and the progress or alleviation of the structural changes, individual differences in language capacity,[1] which is acquired during the life of the individual and perfected by voluntary effort,[2] the difficulty of the intellectual task set, and the recency of acquirement of forms of behaviour in the life of the individual.[3]

The 'diverse facilities' developed by the individual, 'conscious and automatic, (are) all directed to enable him to face a certain situation by the assumption of a distinct attitude.'[4]

Head traced the stages of recovery involving the return of the more recently acquired and more complex types of behaviour in individual patients (vol. i, pt. ii, chap. v).

His classification of the forms assumed by the disorders into four varieties was as follows:

(1) *Verbal aphasia.* Head observed six traumatic cases which fell into this group.[5] 'However fluent these patients may ultimately become, they always find difficulty in pronunciation. . . . Utterance was slow, in short groups of words or syllables each of which was separated by a pause, and speech had a staccato rhythm.'[6]

'All these patients could name objects correctly provided they possessed sufficient mastery of articulated speech. The sounds emitted might be extremely defective, but they bore some obvious relation to the usual nomenclature.'[7] Silent reading was affected, the patient No. 17 said 'I can't read a book to myself. . . . I can get the meaning of a sentence, if it is an isolated sentence, but I can't get all the words.'[8] Writing

[1] Head, i. 220.
[2] Ibid. i. 301–2.
[3] Ibid. i. 429.
[4] Ibid. i. 301.
[5] Ibid. i. 221.
[6] Ibid. i. 221–2.
[7] Ibid. i. 223.
[8] Ibid. i. 223.

shows defects analogous to those of speech—faults of spelling and word formation which tend to be overlooked.[1]

The hand, eye, and ear tests gave further evidence of the interference with internal verbalization.[2] Drawing was not affected.[3] The patients strove to correct their own mistakes.

(2) *Syntactical defects.* The patient 'talks fluently in short jerky sentences, slurring or omitting many of the junction words'. Syntax, rhythm and stressing of syllables are all affected.[4] In severe cases speech was unintelligible jargon. Oral commands in the form of a single word were carried out correctly but not the complex commands of the hand, eye, and ear tests.[5] Printed commands were well executed in all cases and the patients read papers and maps, though sometimes finding difficulty in comprehending what they read. Reading aloud, however, resulted in the same defects as articulate speech, so also did writing, in a lesser degree.[6] Drawing was unaffected.[7] 'General intelligence may remain on a surprisingly high level.' Patient No. 14 'helped himself out of his conversational difficulties by writing single words in a note-book which he carried about with him.'[8]

Head observed three patients who fell under this category.[9]

(3) *Nominal defects.* At first the patient may be dumb, then some power of speaking returns rapidly. 'So far as he can find words they are enunciated correctly and united into coherent phrases.'[10] The most severely affected of this group of five patients[11] could not name one of the common objects in Test (*a*)[12] (p. 98). No. 2 gave wrong names to the colours in Test (*b*), but when shown the names of these colours on printed cards 'began to point to my white coat, to his khaki tie', &c., indicating the appropriate colour correctly; he would describe black as 'what you do for the dead', red 'what the Staff wear'[13], &c. From these phenomena, matched by analogous errors in the rest of the tests and in reading and writing, Head concluded that this disorder of language depends, not only on inability to evoke names, but on want

[1] Head (3), i. 224.
[2] Ibid. i. 226.
[3] Ibid. i. 229.
[4] Ibid. i. 230.
[5] Ibid. i. 232.
[6] Ibid. i. 233.
[7] Ibid. i. 237.
[8] Ibid. i. 238.
[9] Ibid. ii. xiv–xvi.
[10] Ibid. i. 240.
[11] Ibid. ii. xvi–xix.
[12] Ibid. i. 241.
[13] Ibid. i. 242.

of capacity to fit a word, presented orally or in print, to its appropriate object.'[1] 'All these patients could draw from a model and after its removal reproduce the drawing from memory', but with one exception they could not draw (e.g. an elephant) from imagination.[2] These patients could play dominoes, draughts, and chess, but No. 2 could no longer play bridge—'the names of the cards bother me'.[3]

(4) *Semantic defects.* Here the trouble was 'a want of power to combine details into a coherent whole'.[4] Pronunciation, syntax, intonation, and the power of naming were all undisturbed.[5] This was very distinctly shown in the hand, eye, and ear tests, where unlike normal individuals and the other three categories of aphasics, these patients found difficulty in imitating the reflected movements of the observer or the reflected pictures—No. 8 said 'Somehow or another I didn't seem to be able to get the right part of the picture; sometimes I seem to look at one, sometimes at the other.'[6]

Speech and writing tended to be hurried—No. 10 said 'I write very hurriedly so as to keep track of it'.[7] The ultimate significance of matter read[8] and of pictures[9] was missed. 'No. 25 could pass automatically from place to place . . . but as soon as she was compelled to think she became confused. . . . One day she alighted in error at the previous station (to the Marble Arch) on the Underground Railway and not seeing the Marble Arch was completely lost.'[10] No. 8 had difficulty in collecting his things for shaving.[11] Head reported six patients as belonging to this category.[12]

The criticism made of this classification of aphasic defects may be judged by the following. In a discussion on aphasia after Head's 'Hughlings Jackson Lecture' to the Neurology Section of the Royal Society of Medicine, in November 1920, Collier said that the terms 'word-blindness', 'word-deafness', and 'motor aphasia' 'may be fundamentally incorrect, physiologically inadequate, and usually untrue clinically, yet these terms do roughly describe the most obvious features of many speech defects. They have proved useful for teaching

[1] Head, (3), i. 243. [2] Ibid. i. 253. [3] Ibid. i. 256.
[4] Ibid. i. 258. [5] Ibid. i. 257. [6] Ibid. i. 263.
[7] Ibid. i. 261. [8] Ibid. i. 259. [9] Ibid. i. 258.
[10] Ibid. i. 265. [11] Ibid. i. 265. [12] Ibid. ii. xix–xxiv.

purposes and will prove hard to replace.' Purves Stewart said 'This new classification is of the highest psychological value', but he saw no reason for throwing overboard the terms 'psycho-motor aphasia' and 'psycho-sensory aphasia'.[1]

Relation of Clinical Symptoms to Localized Lesions.

In part iii of vol. i of his book, Head recorded his attempt to correlate his four varieties of aphasia with localized unilateral cerebral lesions. There were two instances of verbal aphasia with lesions in the left temporal and Rolandic regions, three of syntactical defects with lesions in the left temporal lobe, two of nominal defects with wounds in the left parietal and left parieto-occipital regions, and four of semantic defects with lesions of the left parietal and right parietal (in a left-handed patient) regions, the supra-marginal gyrus in two cases and the superior parietal lobule in the other two being affected.

In the discussion on aphasia in November 1920, it was generally recognized that the application of Head's tests to other patients by other workers is needed. So far as I know, there is as yet no published report of such an investigation.

In part iv of vol. i, Head correlated his observations of aphasic patients with data obtained by watching the behaviour of Sherrington's spinal and decerebrate preparations[2] and from his own earlier experiments[3] and drew conclusions as to the working of the 'neural hierarchy'.[4]

It is impossible in a limited space to do justice to the thoroughness with which he applied genetic principles and experimental data to every aspect of the problems of aphasia.

APHASIA IN CHILDREN

In proportion to the extensive literature of aphasic defects as found in adults the published work about similar troubles in children is extremely small in quantity, and the greater part of it describes inquiries into the condition of so-called 'word-blindness' or 'visual aphasia'. The *raison d'être* for these has usually been a discovered difficulty in learning to read and write.

[1] Discussion on Aphasia, 413–46.
[2] Head (3), i. 482. [3] Ibid. i. 488–90. [4] Ibid. i. 546.

9. HINSHELWOOD

Hinshelwood,[1] in 1917, gave an account of the examination of 12 children (10 boys and 2 girls) suffering from 'word-blindness'. They had been at school from 3 to 7 years, showed no defects in hearing or vision, were able to learn by heart, to spell words aloud, and make arithmetical calculations correctly. In the absence, however, of any graduated tests it is easy to be misled into diagnosing comprehension and utterance of speech as perfectly normal when there is in fact a defect. (See my patient No. 10, pp. 118–20 and 132–4.)

Hinshelwood accepted the theory of the school of Bastian (p. 88) that 'visual memories of words and letters . . . must be stored up in distinct cerebral areas'.[2] He gave an interesting account of a family in which in the first generation 4 out of 11 children could not learn to read, and in the second 2 out of 6 (nephews and nieces of the 4 previous patients) had the same difficulty.[3] He attributed the defect to imperfect development.[4]

10. FILDES

Fildes,[5] in 1921, published the results of a series of experiments with 26 children aged 9–16 years selected by their teachers as finding reading a very great difficulty.[6] When tested with the Stanford revision of the Binet scale and with the Terman scale only one was found to have normal intelligence, 22 of them being pupils at special schools for mentally defective children. However, Fildes found that 'no relationship existed between the subjects' intelligence quotients and their power in reading. Two of the worst readers were the least intelligent and most intelligent boys'; 'many defective boys with such high intelligence quotients (as 3 of the worst non-reading cases examined) read quite well'. She concluded that the defect is specific rather than general.[7]

The principal tests consisted (1) of the presentation for two seconds each of like and unlike forms on cards. These involved 'right and left or top and bottom reversal' and the subject was required to answer 'Yes' or 'No' according as he

[1] See bibliography. [2] Hinshelwood, 52. [3] Ibid. Chap. iii.
[4] Ibid. 73. [5] See bibliography.
[6] Fildes, 286. [7] Ibid. 287.

judged that he had or had not seen the identical form before. This was supplemented by tests in the reproduction of forms, in 'learning from visual material',[1] 'to test the power of reproducing numbers (in groups of from 2 to 5 figures) presented visually with momentary exposure',[2] and 'to test the subject's ability to recognise words and letters shown under conditions of momentary exposure'.[3]

(2) Auditory tests—learning of digits and sentences (10 to 15 syllables).[4]

(3) Tests of 'the power of making audito-visual associations'—i.e. between a given arbitrary form presented visually and a common name, e.g. 'dog'.[5] This was supplemented by tests in associating words and Greek letters and in learning the names of Greek letters. These tests appear to have been exhaustive in so far as they served to investigate the processes essential to a satisfactory beginning of the process of learning to read. They do not touch the higher level language processes.

The chief conclusions drawn by their author were first that the 'word-blind individuals reveal special difficulties in dealing with material other than words',[6] e.g. the forms above-mentioned.

Secondly, 'the word-blindness is but one aspect of a more general, yet in itself specific, defect in either the visual or auditory regions or both'; e.g. 'the non-readers as a group have greater difficulty in reproducing digits presented auditorily than do the readers'.[7] This is important and consistent with Head's conclusions as to adult aphasics (p. 101), and the view of the unity of the speech-language function put forward in Chapter I of this book (p. 11).

11. BURT

Burt's *Mental and Scholastic Tests*[8] were first issued in 1921. This volume described his extensive investigation into the general intelligence and scholastic attainment of a total number of London school children which approached 5,000.

He showed that 'many young children committed to

[1] Fildes, 292. [2] Ibid. 295. [3] Ibid. 296.
[4] Ibid. 298. [5] Ibid. 300. [6] Ibid. 307.
[7] Ibid. 298. [8] See bibliography.

special schools for the mentally deficient prove afterwards to have been cases of specialised defect, especially defect in linguistic subjects such as reading and spelling'.[1] The primary and practical importance of attempting to find early and effective means of diagnosis of such specialized defects, especially those due to defects in auditory capacity, as in the present research, is obvious.

He described two cases typical of backwardness in reading.[2] To No. 1, a boy of 12 years, are attached the following remarks, significant in view of what has already been said about Fildes's conclusions (p. 107) and in pt. I (p. 11). 'Hearing: acuity normal; perception, discrimination, and recognition of sounds poor, especially if sound long and complex; auditory memory . . . very poor. . . . Enunciation very poor and indistinct.' In Case II 'auditory acuity . . . (was) normal or nearly so' and there was no note as to speech.

12. HINCKS

In 1926 Hincks published a monograph at Harvard on 'Disability in Reading and its Relation to Personality'. She studied 11 children 'very intensively and 4 less so',[3] using three groups of tests: (1) similar to those of Fildes, (2) of handedness, and (3) of eye-movements (photographic methods).[4]

These tests, therefore, like those of Fildes, tended to measure the lower level functions required in the early steps of learning to read.

Handedness was tested because workers such as Ballard[5] appeared to have found a high correlation between left-handedness and speech defects, especially stammering, and it was thought possible by Hincks that an early change of handedness might have resulted in reading difficulty.[6]

Note on Right- and Left-handedness in Relation to Speech.

The problem of the dominant hemisphere (see Jackson, p. 87) and of the incidence of left-handedness is at present very confused. Wallin found that of the whole school population of St. Louis 90·6 per cent. of the children who were

[1] Burt, 266. [2] Ibid. 286–7. [3] Hincks, 5.
[4] Ibid. 7–12. [5] See bibliography. [6] Hincks, 9.

forced to change from left- to right-handedness had no defects of speech,[1] and Ballard now states that he only examined the left-handed children in his survey.[2] It appears probable from Kohler's *Mentality of Apes* that right- and left-handedness is a pre-human characteristic.[3] I gave Gordon's tests of handedness [4] to several of my patients with unsatisfactory results, e.g. No. 1, whom I knew to have some left-handed habits, though never in writing, used his right hand throughout. Yet the precise opposite occurred with the Drever Performance Tests (Chapter VI, p. 112).

Hincks's patients varied greatly in the severity of their reading defect, e.g. No. V read 'exiginicies' for 'exigencies',[5] &c., but apparently had no trouble with simpler words, while No. VI read 'a pog hap a pog' for 'a boy had a dog'.[6] Hincks was not apparently aware of Head's work.

She concluded that there was a high correlation between non-reading and neurotic tendencies, and that reading difficulty is often hereditary. The neurotic tendencies are sometimes *post* and sometimes *propter hoc*. There are also 'certain perceptual variations' as shown by the tests.[7]

Most important in regard to the present research was her discovery of a defect in pitch-discrimination in 72 per cent. of the children.[8] 'This correlation between reading difficulty and defective pitch discrimination is surprising and hard to explain'.[9] In Chapters I and IV of this book I have attempted to show the complete dependence for development of the speech-language function on auditory stimulation over a wide frequency range. It is unfortunate that Hincks made no further investigation of auditory capacity. (She used the Seashore pitch discrimination test.)[10]

13. FROESCHELS

Froeschels alone, in his *Kindersprache und Aphasie* (1918), appears to have investigated the dependence of the speech-language function on hearing. His observations were made, however, without reference to the physical analysis and

[1] Fletcher, J. M., 65. [2] Ibid. 66. [3] Kohler, 32–3 and 36.
[4] Gordon, 316–24. [5] Hincks, 27. [6] Ibid. 33.
[7] Ibid. 89–90. [8] Ibid. 86. [9] Ibid. 87. [10] Ibid. 8.

frequency basis of speech sounds. His 'Case 4'[1] was the most interesting in the present connexion. A boy of 4 years 'of small stature' but with no evidence or history of trouble leading to deafness and with normal tympanic membranes had never reacted to sound or spoken. Froeschels traced the early development of speech in this child, beginning about a month after he had first noticed loud sounds deliberately made in his presence. The record unfortunately ends, in this and in the similar case No. 5[2], before speech proper had begun. But there was clear evidence that this occurred solely as the result of auditory stimulation previously ineffective.

[1] Froeschels, 40. [2] Ibid. 42.

CHAPTER VI

THE SPEECH AND LANGUAGE OF 'APHASIC' PATIENTS

INTRODUCTORY

In accordance with the plan laid down in the introduction to Part II, evidence will now be offered as to the speech and language behaviour of the patients; it will be analysed in order to find whether distinctive features characterize the various groups, and also in the light of the other investigations described in the previous chapter.

Head made it a rule to check the results of his tests of 'symbolic formulation and expression' by tests which were definitely not linguistic.[1]

With my young patients it was specially desirable to find whether there was general mental defect, whether in fact the trouble extended to forms of behaviour not requiring language and not involving reaction to sound.

HEAD'S TESTS UNSUITABLE FOR 'APHASIC' CHILDREN

Head's serial tests appeared clearly unsuitable for use with young children for this and for other purposes. The clock test, for instance, could not be given to a child ignorant of the use of words. All the tests would obviously present different problems to the child learning to speak and understand words in response to special teaching, as compared with the individual who prior to the incidence of his trouble had fully developed language capacity. It seemed better to find a series of graduated non-language tests which preferably should have been previously standardized with a large number of normal children. Results would then be quantitative and more exact.

PINTNER AND PATERSON PERFORMANCE TESTS OF INTELLIGENCE

In 1926 I tested deaf children with the Pintner and Paterson Scale of Performance Tests.[2] These were a composite series with picture completions and form-boards as the chief

[1] Head (3) i. Pt. II. Chap. I.

[2] See bibliography.

features. They were given to approximately 1,000 normal children and a number of deaf children by the authors. The results with the latter have not, so far as I can find, as yet been published.[1]

As used by Mrs. Ewing and myself, these tests seemed to be unduly influenced by the children's previous experience or lack of experience with Montessori apparatus and in the school carpenter's shop.

DREVER PERFORMANCE TESTS OF INTELLIGENCE

In 1927 Dr. Drever (Reader in Psychology in Edinburgh University) kindly gave me facilities for using his 'Performance Tests of Intelligence',[2] then unpublished. These tests partly incorporated those used by Pintner and Paterson, and also introduced a variety of features, e.g. a judgement of weight and several constructional tests. These were less open to the objection just mentioned.

Drever first standardized his tests, tentatively, by giving them to 200 normal and 200 deaf children. He has now completed the examination of a much larger number of children, and the results will be published shortly.

Table H shows the results of giving these tests to my patients.

The previously formed opinion that none of the children was 'mentally defective', but that, on the contrary, supernormal intelligence was consistent with their defect was fully confirmed. In view of the results obtained it was not considered necessary to test all the high-frequency deaf and 'aphasic' patients. The tests require about an hour with each of the younger children. Each child was carefully watched and the results thoroughly examined for tendencies to any constant form of error, but no such evidence was found. The arrangement of Table H is as follows. C.A. = Chronological age of child at time of test. P. = No. of points scored out of possible total 148.

No norms are given above ages 15–16 years, but No. 10's score was obviously inconsistent with a general mental defect. It is noticeable that three of the older patients, Nos. 5, 18, and 9, were the only ones to score a total below their

[1] Pintner and Paterson, 20.

[2] See bibliography.

age norms, while the younger patients obtained more points than normal children of their own age.

No. 1, 8 years old, obtained a normal 13-year-old score.

No. 18 was an extremely nervous and excitable boy, and I do not consider that his score was typical of his intelligence on other occasions.

It appears reasonable to conclude that the defects of the high-frequency deaf and 'aphasic' children are consistent with a 'normal distribution' of general non-language capacity.

TABLE H. RESULTS OF DREVER PERFORMANCE TESTS

	C.A.	P.
High-Frequency Deaf Children		
No. 1	8	105
No. 3	8	92
No. 2	10	85
No. 4	14	(2nd highest score in Test I)
No. 5	14	88
No. 18 (p. xi)	15	83
'Aphasic' Children		
No. 8	8	66
No. 9	13	93
No. 10	17	123

TABLE I. DREVER'S NORMS[1]

C.A.	P. (scored by hearing)	P. (deaf)
6– 7	25	27
7– 8	37	30
8– 9	49	45
9–10	60	52
10–11	71	64
11–12	81	83
12–13	91	86
13–14	100	92
14–15	108	108
15–16	114	112

SPEECH AND LANGUAGE OF HIGH-FREQUENCY DEAF AND 'APHASIC' PATIENTS

The subsequent quotations from my day-to-day records of the speech and language of patients are necessarily brief. I have tried to make them truly representative of similar instances on a number of occasions.

[1] Drever, 43.

The three groups of patients discussed are, first, the high-frequency deaf children, especially No. 1 and No. 2. These two children (with the 'aphasic' No. 7) were the cause of this research being undertaken. Before teaching they were apparently deaf to all speech and, as a rule, to other forms of sound, as has been said; they were also speechless. In Chapters III and IV strong evidence was given that their defect was due to a lack of hearing for notes of high pitch.

The second group, the 'aphasic' children Nos. 7–10, showed marked defects of speech and language, but the audiometer test proved their hearing capacity normal.

The third group have not yet been described. It consisted of two adult patients, aged 40–50, suffering from aphasia.

HISTORIES OF ADULT 'APHASIC' PATIENTS NOS. 19 AND 20

No. 19 was sent to me, in November 1928, with a diagnosis of 'paraphasia'. In February 1928 he had had a 'stroke' and had fallen. A trepanning operation was necessary. When I first saw him he had the scar of the operation wound in the left frontal region and had partially recovered from right hemiplegia—sufficiently to be able to walk with the aid of a stick. Defects of speech and language were extremely severe, and I understand that there had been trouble previous to 1928. This patient has visited me almost daily since November 1928.

No. 20 received a bullet-wound in the left parieto-occipital region in October 1928. He came to me first (for re-education, like No. 19) in February 1929. There was a diagnosis of hemianopsia with partial loss of the temporal field of vision on the right side. Speech and language were severely affected. No. 20 has visited me three or four times weekly since February 1929.

In order to simplify the pictures I have divided the accounts of each group of patients into two parts on the arbitrary basis:

(1) comprehension of language through words heard and read,

(2) use of language in speech and writing.

Head found that though his patients might recover sufficiently to pass superficially as normal individuals, investiga-

tion always showed that the characteristic features of their defect remained through life, e.g. with No. 2, whose 'sister . . . was a trained Froebel teacher . . . (and) dedicated herself entirely . . . to the task of re-educating him'[1] (cf. his No. 8, &c.).[2] I have examined my notes, therefore, to see what features of continuity were presented by each patient during the whole period of special teaching and observation.

A. COMPREHENSION OF LANGUAGE THROUGH WORDS HEARD AND READ

High-Frequency Deaf Children.

My patient No. 1 was in many ways the most striking of the children whose condition led me to make this research. He came to me without any capacity whatever to comprehend or use words, and for the whole period of 5 years, i.e. to the present time, I have been solely responsible for his education. In the first place response to speech was brought about by the use of lip-reading, though from the beginning a simultaneous effort was made to teach the patient to listen to words. This is the method which has been used to teach all the high-frequency deaf children. There have been two phases in the education of these children (this necessarily applies more strictly to my private patients than to those who had to be taught in a class with deaf children at school).

(*a*) Acquisition of language partly by ear but chiefly by lip-reading (+ touch sensations).

(*b*) Acquisition of new words, chiefly by ear, lip-reading being only used as an aid.

With No. 1 the change from (*a*) to (*b*) began in about the thirteenth month; e.g. there had been a thunder-storm overnight; in the morning he used the words 'thunder' and 'lightning', which I had not taught him, and articulated them correctly. These are words which it is extremely difficult to learn by lip-reading without the help of a trained teacher; No. 1 had 'picked them up' at home and uttered them with the natural intonation which is acquired through the ear alone. To-day oral teaching is often given

[1] Head (2), ii. 30. [2] Ibid. 108.

with the patient in such a position that he cannot see my face unless I wish him to do so.

With all the high-frequency deaf children the effect of much or little reading is very marked. This is, of course, quite intelligible on the basis of the evidence in Chapters III and IV—that as distance from the speaker increases and intensity of the sound decreases, only fundamental (laryngeal) tones of the voice tend to be heard in quiet conversation. The effect is one of indistinctness which before teaching clearly amounted to complete unintelligibility. Lip-reading, on which the patients rely chiefly during phase (*a*), is more distinct but still only gives impressions of about 50 per cent. of the movements productive of articulate speech. Reading, by contrast, once acquired and if much used, gives the high-frequency deaf child words in a distinct and complete form. I began to teach No. 1 to read in the eighth month. In the twelfth month he was reading the numbers on the trams for his own amusement. Since then he has developed a passion for reading any book or daily paper that comes his way, as evidenced by his spontaneous speech during the last two years, which has contained references to the armour worn in the 'twelfth and thirteenth centuries' (traced to his sister's history book), an old lady of 121 reported by the *Daily* —— as having a 'great-great-granddaughter and to be still able to work', the exact age of Queen Victoria at her accession, &c.

No. 2, on the contrary, whose education has been much interrupted, has not yet learned to read for pleasure and her speech and language development has been less rapid.

Comprehension that everything has a name, which comes to the normal child at the age of from 18 months to 2 years, began to dawn on No. 1 in about the second month of teaching, when he began to say 'Ti-ta', pointing to objects, and sometimes spontaneously to watch my face in order to lip-read. With No. 2 spontaneous attempts to comprehend words began rather later.

As in the case of normal children, there has been clear evidence that comprehension of language has always been ahead of the use of words in utterance and writing. All the children were able to make a constant response to words taught and spoken by the teacher (e.g. they could point

out objects named by the teacher) several weeks at least before they could speak any words. The length of this interval of time was about a month with Nos. 1 and 2.

In Part I evidence has been given to show that the comprehension of words heard by these patients depends primarily on a constant factor—their distance from the source of sound and its intensity. That No. 1 can now comprehend speech heard of a standard of development at least normal to his age, provided that the intensity of the sound is adequate, is evidenced by the following quotations. I read a story to him in a fairly loud distinct voice, sitting close behind his chair: 'By this time the fawn had grown two little antlers. He was so proud of them that he damaged many saplings in order to see how strong they were. . . . Sometimes when he ran out of the plantation into the pine-wood the wire fencing would catch and hurt them. This spoilt the shape of his horns and they were crooked all the rest of his life. . . . He was feeding. . . . when the sound of footsteps made him crouch very low. . . . Presently a pony came down the track with a man by its side. Tied on its back was a red deer.' A week later he wrote the following unaided reproduction of this story: 'The little fawn had a pair of horns which he was so proud of, and damaged a lot of small trees to see how strong his horn was. He went to the woods to play, when he heard a footstep of a man and a pony with a stag on it. He ran away.'

I asked, 'How did the fawn know they were the footsteps of a man and a pony, and that the pony carried a stag?' He replied, 'He smelt it.'

The excellent conversational powers of Nos. 1 and 2 are illustrated below.

In the comprehension of language read the capacity of the high-frequency deaf patients appears to depend chiefly on the amount of teaching and partly, as with normal children, on individual preferences and environment.

No. 1's passion for reading has already been mentioned. In the eighteenth month he would read the advertisements in the town. In the second year he insisted on having a weekly paper. By the beginning of the third year he was sufficiently advanced to get out his father's atlas on his own account and to say, after looking through it, 'There is no

Never-Never-Land, because I look in the map-book on every page and did not find it.' He had been reading the story of 'Peter Pan'.

In March 1929 I gave him Burt's Reading Tests.[1]

In the 'Graded Directions Test (Comprehension)' he was able to carry out correctly the printed instruction, 'Turn with your face toward the window before you read the rest of the card. When I tap, walk two steps away from me. When I tap again, raise your empty hand. When I tap the third time, do nothing. At the fourth tap bring me the card'.[2] He gave a correct answer to the following:

> 'The greenest buds of May,
> The brightest flowers of June,
> To me are never so gay,
> As a brown October day,
> With its golden sheaves,
> And its crimson leaves,
> And Autumn tints of decay.

Which month does the writer think the most beautiful—May, October, or June?'[3]

For a moment he was puzzled, saying: 'What do they mean by that? Wait a minute, it partly (I see it partly?)—May, June' (looking down the page), then emphatically 'October'. This performance was equal to the better 50 per cent. of the London schoolboys at nine years.[4] His own age was 9 years 6 months. It was a surprisingly good performance in view of his limited language experience. No. 1 is undoubtedly the most advanced of the group for his age. No. 2 at 11 years obtained a bare 7-year-old score. Her education, however, had only been continuous for 1 year 9 months.

'Aphasic' Children.

Of the three patients of school age who presented marked defects in speech and language but were found to have normal auditory acuity, No. 10[5] was perhaps the most interesting. When I first saw her at the age of 14 years she answered all questions and talked about her school and home, games, the pictures in the Art Gallery, &c., in a manner normal to her

[1] Burt, 269–83, 339–53, and 399–402.
[2] Burt, 347. [3] Ibid. 348. [4] Ibid. 400. [5] See p. 40.

age. At this consultation I thought that reading and writing only were affected. However, during the course of lessons, I found signs that her vocabulary was limited, e.g. she said she was going to take her watch to the 'watch-shop'.[1] At first it appeared that this might be due to her having lived an open-air life at home and to inability to read. Her general mentality was clearly not defective—she could discuss agricultural matters with interest and knowledge—speech appeared normal (cf. p. 132). She came from a cultured home. Later I found evidence of marked incapacity in response to speech heard. In repeating a story read aloud to her phrase by phrase she made the following mistakes. The correct word is given first in each case, then her version:

> admit, a'mit (twice); snarled, star; reputation, recreation; support, report (susport).

On another day similar errors were made in the 'Fox and Goose' story from *Aesop's Fables*:

> provided, pervided; agreed, regreed; ordered, order; obliged, compliged; consent, content; with as good grace as he could, with good grace as he could; observing, preserving.

On another occasion 'When Diogenes received a visit in his tub from Alexander the Great' was rendered 'When Diogenes received the visit in his tongue from Alexander the Great', and 'according to the ancient forms of royal courtesy' became 'according to the ancient cords of royal courtesy'. These results were not affected by the intensity of the speech-sounds she heard. It must be emphasized that there was no defect in articulating words which had become part of her own vocabulary and which she used in spontaneous speech.

She was completely unable to read with comprehension before teaching by special methods began. In April 1927 she read the whole of 'Sapper's' novel, *Bulldog Drummond*, for her own amusement, in about a week. She was able to answer questions about the story correctly, and said that she

[1] When I tested her with the word 'jeweller's' she had the greatest difficulty in repeating it correctly.

had occasionally asked help with words she could not recognize. There was no difficulty, from the first, in recognizing visual forms. She reproduced six Greek characters correctly after a minute's exposure. But even two- and three-letter meaningless syllables still presented difficulty, as shown by the following test of reading aloud. The syllables on the upper line of each pair are those on the test sheet, the lower her version:

1.	AM	OB	GOT	PUT	TUB
	AM	BO	GOT	PUT	TUBE[1]
2.	DEB	SIT	LOG	MET	REP
	DEB	SET, SAT, SIT	LOG	MEET MET	REAP
3.	JAG	DAP	NOG	MEN	WEEK
	JAR, JAG	DAMP, DAP	MOG, NOG	MAN	WEQ

On the same day she was also tested in reading sentences. Her mistakes are in brackets.

1. You must pull hard and the doors (door) will come open.

2. That is what comes of not having the garden weeded (correct).

3. Tin was found in that part of the county (country) and mines were started by the owners (orders, owners).

4. As the air darkened (darkens, darkened), it turned (turns, turned) gradually warmer.

5. A grey-haired man entered the drawing-room (correct).

6. A broad piece of wooden board (boarded, board) drew their attention.

This was a great improvement on her attempt to read a simple story aloud when I first saw her. Then the result was a hurried meaningless jumble of syllables.

Patients Nos. 8 and 9 were able to understand what was said to them so long as the words were not unfamiliar. Their vocabulary was extremely limited for their age. But not having used a graduated test for this I propose to reserve discussion of their defect in hearing speech to the second half of the chapter. As regards reading, when No. 9

[1] 'I was not thinking about the little "e" at the end.'

first came to me in October 1926 at the age of 11 years he could not read all words of three letters correctly, in spite of having begun to attend an elementary school at the normal age. His speech was so incorrect that his teacher could not judge the accuracy of his attempts to read. There was no known physical defect or history to account for his condition. Adenoids and tonsils had been removed about 1922, but there was clearly some other cause for the defects, since speech remained unintelligible four years afterwards. There was no difficulty in recognizing letters written or printed. During the course of lessons with me—he has only been able to make 70 visits in $2\frac{1}{2}$ years—the reading capacity has advanced concurrently with the improvement in speech. There is definite evidence that his whole education, as is usual in such cases, was 'held up' by the speech-language retardation and consequent inability to read. As the result of the lessons his speech is now completely intelligible and is rarely marred by defects in articulation. He remained at school until July 1929, and his teacher reported that he made good progress latterly in school subjects.

No. 8 was beginning to 'spell out' words from a phonetic reader when I first saw him. He now answers questions correctly about a simple story which he has read.

No. 7's early condition was described in Chapter I. The discovery during this research of the condition described as high-frequency deafness came as a complete surprise; but the results of examining No. 7 by the methods described in Part I have been even more unexpected, and they appear to illustrate the diagnostic value of the new tests of hearing capacity in a most convincing manner. This is more strongly the case since Mrs. Ewing has been able to supply me with detailed and verified notes of this patient's condition on entering the Royal Schools for the Deaf, Manchester, at 4 years 9 months, and of her early development in response to methods of teaching similar to those described earlier in this chapter.

She was sent to the school with a diagnosis of total deafness of congenital origin. There was no known history of injury or serious illness. She was small for her age and had a small-sized head. Her whole behaviour was that of a child deaf to sound. She was dumb. Her behaviour, however, was

intelligent. She had not been at the school two years before she showed a unique capacity for mothering the younger deaf children in out-of-school hours. She was more than usually capable of looking after herself. Her movements were quick and accurate, and she did not drag her feet in walking like deaf children. At first she was markedly 'bird-witted', but she learned to watch for speech and to lip-read a few words (her own name, names of children, nurses, toys, &c.). Then she began to babble, but made no attempt to utter any particular word.

In the third month after admission she, with other children, was asleep one night in the dormitory. In schools for the deaf a dim light is usually kept burning in the children's bedrooms, but on this occasion the electric light had just fused. A teacher and a nurse were talking in the room and No. 7's name was mentioned. She sat up and called out, though unable to use words. Next day she was tested while playing in company with the other children; she was able to respond to her name (not to the names of the others) when her face was covered with a pinafore.

From that time her capacity to respond to speech heard rapidly improved. Her use of speech and language steadily developed. By the end of her second year at school her comprehension of speech heard had reached the '2nd period' (Chapter I, p. 6), and she used words as her best means of communication, helped out by gesture. But it must be noted that throughout the first four years of school life she could not learn new words for the *first* time through hearing—she had to lip-read them: e.g. two visitors came to the school, whose names were Mrs. Bothwell and Mrs. Remmington. She could, after having been introduced to them (i.e. by lip-reading and hearing) point to each when asked by the teacher standing behind her, but she could not repeat either name until she was allowed to imitate it directly by watching each syllable and so building up the word.

At this stage there was still evidence, on analysis, of difficulty in comprehending speech by hearing alone. To-day a similar defect is discoverable in her language comprehension on close investigation (pp. 123–4 below).

At 16 years she could converse freely with friends and strangers, read and write fluently, and go shopping by herself like any normal girl of her age. She could gain new words by ear alone.

She has always been very highly strung and sensitive. At the age of 14 years she had an attack of chorea.

In April 1929 she was 22 years old. It was obvious that she was a patient whose symptoms and history might yield important data. She willingly agreed to come from Wales to Manchester for a day. She was now employed as a waitress, at a good salary, in a small hotel. She had been two years in her present situation, and her employer was very sorry that she expected to leave shortly in order to get married.

Her behaviour was a striking tribute to the education she received at the Royal Schools for the Deaf, Manchester. Her parents were very poor and illiterate. Her father was a working saddler aged 68 years. She contributed all her wages, except 1*s*. weekly and the cost of her uniform, to keep up the home. Her speech in conversation, table-manners, &c., were those of an educated girl from a cultivated environment. Superficially her speech and language would have passed as completely normal.

Her auditory acuity, measured by a most thorough application of the audiometer test (Chapters III and IV) was normal (Table B, p. 40). 'Descending' as well as 'ascending' order of intensity steps was used. But a marked feature of the results was the improved response (10–20 per cent.) after 3–6 repetitions of any given frequency. For instance, two initial responses to 4,096 v.d. were at intensity step 20. But on returning to this frequency after the conclusion of the whole test a reaction took place at 5 and at 10. Auditory repetition of numbers was not apparently affected by the loudness of the speech sounds I used as stimuli (contrast Nos. 1, 2, &c., p. 64), but there were mistakes with numbers above 12, e.g. '32' for '22'. The numbers confused were definitely not those which are difficult to the high-frequency deaf children. She conversed freely about her fiancé, her work, her school-life (with accuracy of detail in regard to events even 16 and 17 years ago).

A graduated test of her reading capacity, however, gave further evidence of a difficulty still remaining in comprehension (Burt's Reading Test No. 4[1]). Her performance was considerably inferior to that of No. 1 (aged 9 years, p. 118). She could read all the words as single words (proved by reading aloud) but the meaning of the complete directions escaped her. Given the printed instruction, 'Open my book at page 8. Put the pencil between the leaves of the book. Shut the book. And then say to me: I have done what you asked'.[2] She opened the book at page 28 and ignored the part from 'Shut the book' onwards. No. 1 carried out this test correctly. She could not grasp the meaning of the verse of poetry (p. 118) which No. 1 understood. The test was carried out correctly by the better 50 per cent. of London school-children aged 7–9 years.[3] The same difficulties were shown in other parts of it, which were of an equal order of difficulty, though to '"So the shepherd brought his flock to the market; and the animals were sold to make mutton, after their wool had been cut off to make cloth". What kind of animals were they?'[4] she gave the correct answer. In Burt's Reading (Comprehension) Test No. 5[5] in which she had to answer 20 questions about a story of some 200 words which she had just read, she made 12 correct replies—the average score for the London schoolgirls of ten years.[6] In the 'Graded Vocabulary Test' No. 1[7] she reached the same standard.

The type of error made will be discussed in the second part of this chapter. She says that she reads little, and this taken with the general conditions of her life might tend to minimize slightly the significance of the findings just given. But further evidence that comprehension of speech heard involves difficulty was given by the patient. She says that she cannot follow every one's speech without conscious difficulty, and that she cannot recognize the words of the 'announcers' or of singers when listening to the 'loud-speaker' at home. She has some difficulty with 'change' and other money calculations.

[1] Burt, 346–9. [2] Ibid. 347. [3] Ibid. 400.
[4] Ibid. 347. [5] Ibid. 351. [6] Ibid. 402.
[7] Ibid. 340.

Adult Aphasic Patients.

No. 19 (p. 114) is an educated, much-travelled man of property. Up to the incidence of his trouble he controlled his own works. To-day he makes every effort to keep in touch with the progress of the business. With this patient knowledge of having made a mistake in any task had a strongly depressing effect, and at first would prejudice the results of any further efforts at a given visit. Serial tests as such have been impossible, but nevertheless evidence has been collected as to his psychological condition. Audition and vision were unimpaired, the former even perhaps more acute than in the average adult.

The outstanding feature in November 1928 was the condition of almost complete speechlessness (pp. 136–9). There has, however, been evidence of great improvement in speech-language capacity during the period of re-education, November 1928 to March 1930.[1] Capacity for mental concentration without undue fatigue has also increased. At first he was unable to attend continuously to any task involving speech and language for more than about five minutes, now fifteen minutes is not too long as a rule.

He seemed to understand all that was said to him at his early visits. He would reply non-committally 'Quite', whether he comprehended what had been said or not. His general powers of thought were extraordinarily good. He was determined to make himself independent of any help in personal matters such as dressing. Gradually he contrived to do everything for himself, although the right hand was still relatively useless for grasping.

On close investigation defects of comprehension of language heard and read, similar to those which characterized his speech and writing, were clearly proved, although as indicated in Moutier's description of the aphasic patient (p. 92) he usually understood 'Clearly', as he said himself, if spoken to rather slowly, in familiar words, and not too long sentences. Simple tasks of pointing out objects named were carried out correctly, but even in December 1929 he still scarcely trusted

[1] Patients Nos. 19 and 20 have consistently progressed since the accounts of them given in this book were written. No. 19 now shows only relatively slight traces of aphasia.

himself to have understood the day and date mentioned for an appointment. Sometimes he would attempt to repeat the day and name it correctly or incorrectly in a questioning tone. Sometimes he indicated, more often by word than gesture as at first, that I was to write it down.

From time to time in conversation (he was usually able to make appropriate short verbal answers) he would repeat a word said to him with the intonation and facial expression of one completely confused: for instance, I was describing the colour-scheme of a room and said, 'The furniture was green and gold'. He said: 'Gold? What?' I said, 'Like your ring. Like your watch.' This apparently conveyed nothing. I then said, 'Like those tulips (pointing), they are gold.' He then said 'Gold', and pulled out his watch.

The same trouble occurred in silent reading. On one occasion he was looking at an illustrated paper when I entered the room. Pointing to a photograph of some blue-jackets being taught to groom a horse, he indicated the words 'vocational training' beneath and said, 'Voice?'—evidently confused by similarity in the visual forms of the two words.

Head has remarked the factor of complexity in the reactions of aphasic patients. Its influence is very marked with this patient. In November 1928 I used to read short simple stories aloud to him, which he seemed to enjoy. One day he indicated very emphatically, chiefly by gesture, that he did not wish the nurse, who still accompanied him at that time, to carry out this form of practice at home as I had recommended, but pointing to me he said: 'Clearly: Oh! distinctly.' I gathered from the whole incident and from his other efforts to explain that he considered that the nurse read too fast without long enough pauses and somewhat indistinctly. At that period he would have no humorous stories read or told to him. Now he goes to the theatre ('straight' plays and musical comedies) and enjoys it.

No. 20 (p. 114) was also well educated and travelled. He was in an important position before receiving his wound. His wife reported that he had always been fond of reading. His reactions to speech and language bore this out, as with patient No. 1. In view of Von Monakow's investigation of

the phenomenon which he describes as 'diaschisis', it was presumably probable that No. 20's condition was at first complicated by non-permanent features. The defect when I first saw him was very marked, but though his speech was most confused and sometimes unintelligible, he was able to carry on a conversation and never showed signs of fatigue as the result of mental concentration during the sittings. Asked to point out objects which I named aloud he never had any difficulty. But asked to set the hands of a 'clock-face', as in Head's tests (pp. 98–9) he made the following errors: I said 'Quarter past one', he set the hands at 'Quarter past three'; I said 'Twenty past one' he murmured to himself 'Twenty-one', and set the hands at 3.20. Of reading, he often spoke himself to the following effect: 'I get on all right till I reach some word, then I get bothered and confused.' As the result of teaching he was soon able to read a book with a good understanding of its meaning.[1] But this matter touches on the problem of reading aloud, which I propose to analyse later in this chapter. He was at first evidently uncertain as to the accuracy of his own attempts to speak and even when his utterance was correct would try to repeat the word again, often more than once and incorrectly. After writing the correct answer to a simple addition sum presented to him in writing, he remarked: 'Mind you, I don't feel sure.'

In this part of the chapter I have endeavoured to give carefully selected illustrations of difficulties of a receptive or cognitive type. The interdependence of the receptive and expressive powers, of the cognitive and conative aspects of each complete act of behaviour, was nowhere more fully illustrated than by No. 20's efforts to 'set the clock' and his spontaneous attempt to repeat aloud what was, for him, at that time, a difficult instruction.

[1] In Burt's Test 4 (cf. Nos. 1 and 7, pp. 118 and 124) he could not, in March 1929, carry out the instruction, 'Put two more pins into the box and one near it on the table.' (Age 6.)

B. USE OF LANGUAGE IN SPEECH AND WRITING

High-Frequency Deaf Children.

The development of speech in the high-frequency deaf children, in response to teaching, appears to pass through the same stages as that of the normal child.

The records of No. 1 clearly illustrate this:

(1) 'One word sentence' period, second to sixth month of teaching:

Nanny = calling his nurse.
Gome = go home, when his coat was put on.
Pom-pom = on seeing the motor-car.

(2) Words combined, beginnings of syntax, increase in vocabulary (sixth month to fifteenth month):

6th month (of teaching): 'Please coat.'
9th ,, 'Jump high', 'Joan school'.
12th ,, 'Pussy up'tair home pussy hide'.
13th ,, 'Mummy, Daddy, Stutter (himself) Joan pom-pom (=motor-car)'.
'Mouse upstairs stick'—(he had seen tame mouse run up a ladder to its sleeping compartment).
14th ,, 'Lamp gone Joan cry' (Electric light had failed the night before and his sister had cried).
,, ,, 'Stutter home black blue red yellow a lot' (=I have a lot of playing cards at home).
,, ,, 'Like pussy' (=just like).
,, ,, 'Small horse big horse' (comparison).
,, ,, 'Daddy, Mummy gone away, motor engine boat' (=i.e. by motor, train, and steamer).
,, ,, 'Where black bag?'
,, ,, 'Handkerchief, show it Granny'.

By the end of the 14th month No. 1 was able to talk intelligently for a minute continuously, describing himself and his family going away for a holiday, having lunch in the train and meeting their relations.

15th month 'Polly not red tail' and 'not that one'.
16th ,, 'Ina come upstairs'.
,, ,, 'Nancy no, Joan's, put it down, (=No Nancy, that's Joan's, put it down).

17th ,, 'Ina boat gone sea' (Ina has gone in a boat over the sea).

'Come Mummy garden quick'.

The above quotations illustrate the development of No. 1's grammatical construction and its close similarity to that of the normal child as described by Stern and Jespersen.

(3) Inflections appeared about the 16th month as shown by the words 'Joan's' and 'gone'.

(4) Compound sentences with subordinate clauses began to appear about the 27th month with:

'Make a big big snow-ball, what for?'

'Ruth and Tommy have got a brown mouse (which) go (goes) upstairs into the other box. In the straw (the) mouse has (a) stool and eat (eats) toast.'

'How many days (will it be before the) snow blow (=will go away)?'

The difficulty for these children in hearing consonants and unstressed words and syllables has already been described in Chapter IV. It seems clear that this is the reason why the high-frequency deaf children have been slower in beginning to use sentences completely correct in syntax and inflections than in enlarging their vocabularies and the length of the 'word-collections' they utter: this too in spite of strenuous efforts made by me to correct these deficiencies.

Otherwise the 'mistakes' in their speech have been those of the normal child:

elision, 'i'-ream' (ice-cream), 'up-tair' (upstairs);

change of sound (complicated by lack of hearing for consonants), 'putch' (push) 'very night' (very nice), 'kilk' (kilt);

metathesis, 'nets' (nest), 'hog-hedge' (hedge-hog);

re-duplication: 'umbillella' (umbrella), 'ellelli' (elephant), 'funnunnel' (funnel).

No. 2 used what seemed like jargon for a time, but that it was not true jargon but imperfect imitation of what she heard, like the normal child's 'sollibub' (suburb)[1] and 'kather' (father)[2] is proved by the disappearance of these symptoms to-day. The explanation of the apparent

[1] Stern, 160. [2] Ibid. 156.

temporary abnormality seems to have been the fact that she is a naturally sociable and expressive child, she had had sufficient teaching to give meaning to some of the louder sounds (chiefly vowels) reaching her with adequate intensity from the speech of her environment; she had begun to learn to talk and then teaching was for a time almost completely withheld. The result was that she made the best use of her speech-language function so far as it had been developed.

To-day the speech of patients Nos. 1 and 2 increasingly approximates to that of normal children of the same age. Indeed No. 1 talks so freely and distinctly that it is doubtful whether an average listener, hearing him speak for the first time, would notice any abnormality.

The high-frequency deaf children, except for No. 4, have always used a normal range of laryngeal tone, i.e. intonation. The following examples illustrate the speech of these children. No. 1 (March 1929), discussing a Meccano model he had designed himself, 'I've forgotten the push-button. There's a battery underneath, for how could it light itself if there was no battery. They are marked (on a diagram he drew for me) where they are. That easily tell(s) you where it is (they are)',

No. 2 (March 1929), was naturally less advanced, in fact only just entering the syntactical from the 'word-collection' period: 'Mother and Daddy gave me my birthday some nice clock'. Myself 'Where do you keep it?' No. 2, 'On the bedroom and in Nursery.' Also '(Last Saturday) Martyn and I went out on (a) ship with Uncle Jim', &c. Some of the vowels and consonants still differed slightly in sound from those of the normal child, but speech was definitely becoming more and more normal.

Reading aloud, with these and many patients, is as much a test of speech capacity as of the capacity for comprehending language through the eye. With none of the high-frequency deaf children was there an unbroken continuity of natural development of the speech organs from screaming to babbling and babbling to direct imitation of words. There was a break in this succession before teaching began at the age of 4–5 years. Yet tested in March 1929 with Burt's Reading

Test No. 3 (Speed)[1] patient No. 1 (aged 9 years) read 122 two- and three-letter monosyllables in one minute, and No. 2 (11 years) 75, average performances for normal children of the same sexes, aged 13 years and 8 years respectively. It must be remembered that $4\frac{1}{2}$ years or more of auditory experience had been missed in each case.

In the Graded Vocabulary Test No. 1,[2] in which the subject has to read aloud words of difficulty graduated from 'to' to 'phthisis' No. 1 reached 'events, motionless', with only three errors, he tried 'economy' 'Icony, Help! must I be able to say that?' and picked out 'universal, circumstances, glycerine, emergency' and read them correctly. He said 'Emergency, I know what that means, "emergency door" in ——— buses.' The score was equal to the normal London school-boys' at ten years. No. 2 has had far less teaching, is one of a family of four, and so far has spent nearly all her time at home in active play or helping with her younger sister (in doing this she shows distinct capacity and shrewd commonsense). Nevertheless, she reached 'village, nurse, quickly, return' correctly and obtained a score equal to the average normal school-girl's at age six to seven, and equal to the average mentally deficient children of 13 years.[3] In March 1928 she was able to read few words of more than 3–4 letters.

The powers of these children in writing, to dictation or spontaneously, in letters or the reproduction of a story, are in proportion to those already described for speaking and reading. They show no abnormalities in learning arithmetic or to tell the time.

With the other high-frequency deaf children I have not had the same opportunity for making continuous daily records, but from very close attention to their speech and language behaviour during the tests recorded in Part I and for the period November 1927 to March 1929, I feel justified in concluding that their speech and language development is in proportion to the amount of teaching they have as yet received and their individual aptitudes, and that such 'errors' as they commit are as equally representative of normal growth as those of Nos. 1 and 2.

[1] Burt, 344. [2] Ibid. 340.

[3] Ibid. 400.

'Aphasic' Children.

The speech of patient No. 10, in conversation, was characterized by no abnormal symptoms. Yet, as I have already described, the defect was gross directly she attempted to read aloud or to repeat after me words with which a girl of 14 years, from a similar or less favourable social and educational environment, is as a rule undoubtedly familiar (cf. standards of Burt's Reading Tests, as illustrated in the preceding pages). Her spontaneous attempts in written language manifested her trouble in a most striking manner, as shown in the following quotation from a letter written in June 1926, before she came to me. 'I show you and Miss X- had a nice time together? Will you where the nice long woolen cost to fetch us, I is caal stick that done this. We orther settle for a wh. Sone of the farner round her have got there hay in and lot of it is cut. . . At co ohary this week or we made short.' The meaning, so far as I can construe it, was 'I'm sure you and Miss X- had a nice time together. Will you wear the nice long woollen coat to fetch us? . . . (The next two sentences I cannot understand) . . . Some of the farmers round here have got their hay in and a lot of it is cut. . . At cookery hour this week (note the transposition of words) we made shortbread.'

It seemed clear that improvement in written language could not be expected before the patient was able to read. With the normal child and adult ability to comprehend (therefore discriminate between, remember and recognize) language heard is always in advance of the power to use words in speech and writing—'The inferiority of what can be said compared with what can be understood is a peculiarity which continues even in adult years—for every one of us uses a much smaller vocabulary of words and phrases than he hears and understands in lectures, &c.'[1] It is to be expected that the individual's vocabulary will be less extensive than that of his whole social environment. For patient No. 10 reading would open up a new source of language with all the completeness of the printed page.

There was no difficulty in recognizing isolated letter forms, as was proved by the experiment in discriminating between, remembering, and writing, six Greek characters, after a

[1] Stern, 145.

minute's exposure. (In fact it is doubtful whether difficulties on this level can be described as speech-language defects: they involve no act of 'symbolic formulation and expression' any more than the process of learning to fit together the parts of a safety razor.) Every specific response to a specific situation in a creature capable of variable reactions involves both analysis and synthesis. In reading, the analytic features include differentiation between the words on a page and selection of one word (in the early stages of reading, later of course larger groupings), as the object of attention. There is also discrimination between the various letter-forms. When words are polysyllabic the power to analyse words into syllables is required. The synthetic aspect includes, in the first steps of reading, i.e. in reading aloud, learning to react with the auditory-motor speech-language habits already developed, to visual stimuli. It seems clear that there is a fitting together of words previously heard (and their meaning) with the process of differentiating between, recognizing, and remembering, the conventional and arbitrary letter-forms, whether first presented singly or in groups.

With patient No. 10 difficulty in reading and writing was shown in both analysis and synthesis:

1. Improvement in reading was greatly assisted by use for a short time of a piece of cardboard with an opening in it, all the words on a page except one being thus covered up.

2. While single letters were not confused (see above, an alphabet test also confirmed this), the increased complexity of groups of them resulted in confusion of groups with specific similarities in appearance, e.g. OB was read BO, &c. sometimes also in mistakes on the lowest level, e.g. the confusion of 'm' and 'n' as shown by 'sone' for 'some' in the extract from her letter above. Examples of this feature constantly occurred.

3. Similarity in the sound of words was responsible for many errors in reading and writing, e.g. 'show' for 'sure' in the extract from the letter; again many instances could be quoted.

4. Difficulty was experienced on the higher levels—analysis of words into syllables, synthesis of the syllables back again into the complete word, synthesis of words into

phrases and sentences and of the underlying meaning; this was to be expected. (Compare Jackson on the 'proposition', Chapter V, p. 85.)

Progress resulted in response to teaching, e.g. this extract from an unaided reproduction of a story she had read (April 1927): 'There was once a Brahmin he was walking along the road when he saw a tiger in a cage. The tiger *ask* (asked) the man to let him out. The Brahmin said he would if he would not eat him up, &c.' In December 1928, she wrote me a letter from which I give extracts: 'Dear Mr. Ewing, Mother wrote and told me you would like to see me, so I *though* I would write and tell that I would very much like to see you. It seems a great long time since I met you. I have been meaning to write all term but I have been rather busy. What a great *nouiance* the snow is, we were having a cup match final, but it snowed so it will have to *waite* till next term. . .'

As regards the other non-deaf patients, No. 9's (pp. 39, 120) difficulty on analysis was found to be of a very specific nature: he could not articulate correctly any of the consonants 'T', 'C' or 'K' (*c*all, *kick*), 'F', 'S', or 'R'. In the consonants 'T', 'K' and usually 'P' the velum was not elevated as is necessary for correct articulation. It was scarcely elevated in the vowels.

The problem therefore arose as to whether perception was imperfect. But this was not so. Both crude hearing (Table B, p. 40) and the capacity to recognize these consonants when I made the appropriate sounds behind his back, at any degree of loudness or softness, and to point them out on a printed sheet, was perfect. Nevertheless later experience showed that even when aged 14 he was unacquainted with such words as 'tusk' and that the combined effects of imperfect speech and less than average general intelligence had resulted in very backward speech-language development as a whole. Elimination of the specific defects through special teaching has led to marked progress both linguistically and educationally (pp. 120–21). His mother remembered with certainty that speech was very late in beginning. I suspect that either 'babbling' or imitation or both began late and took place to a limited extent. From his rather small size, especially as regards the head, it seems possible that some

factor inimical to general development was present during the first years of life. His mother reports that he was delicate from birth.

No. 8's speech at 8 years was characterized by mistakes such as normal children make in the second and third years of life. Extract from spontaneous reproduction of the story 'The Three Bears': 'Oncetime see Glandma'—(his mother 'No B-, that's about "Little Red Riding Hood"'). The patient 'Onetime Goldilock (Goldilocks) seeacottage (saw a cottage), liffupalatch (she lifted up the latch), walkin (she walked in), seethleebowlporridge (she saw three bowls of porridge)', &c. In the same way 'bissn sis' meant 'Robinson Crusoe'.

There was a remarkable use of the 'one-word sentence' in speech when comprehension of language and other forms of mental development had left that stage far behind.The mistakes in imitation were those of elision, e.g. liff for lift, bissn for Robinson; change of sound, 'Glandma' grandma; and contamination, 'sis' from a combination of Rob*inso*n and Crusoe.

Crude hearing was normal (Table B, p. 40) and as with No. 9 vowel and consonant sounds could be discriminated by ear alone. Comprehension of language showed no superficial defect. The patient was not mentally deficient as shown by the Performance Test results (Table G, p. 113) and other non-language behaviour. But his vocabulary (comprehension of words) did not extend to such words as 'snored' and 'queen'. A younger brother aged 4 years spoke normally. The patient's mother reported that at 3 years he could only say 'Mummy' and 'Daddy' and that he has always shown unwillingness to talk.

Neither with No. 8 nor No. 9 was there difficulty in learning by heart. With both, very marked progress has been obtained, first by teaching the patient to repeat the consonants correctly alone and in syllables, drawing attention to the movements of the speech organs required, and then by intensive teaching in reading.

With No. 8 there is evidence therefore of retarded development in speech and language, but the general character of the principal features of the defect contrasts with the specific deficiencies of No. 9. With No. 8 at 8 years articulation of

particular vowels and consonants was more correct than with No. 9 at 12 years. It is suggested that the trouble affects a higher level and was possibly less severe. The good progress of both patients seems to indicate that the trouble took its source in a temporary stunting of development; but final proof can be obtained through time alone.

The striking results of the education of No. 7 by special methods have already been described. In April 1929, six years after leaving school, she spoke with scarcely a trace of defect (cf. pp. 121–24). Her letters were perfect in expression of thought, choice of words, spelling, and contained few grammatical errors. She spoke quietly but with normal intonation, phrasing, vocabulary, and syntax. The following slight points were noticed in the course of a long conversation: (1) There is a tendency, not invariable, to omit the ends of words, e.g. 'I've bee*n* nurse to a doctor's children,' 'I slipp*ed* downstairs' (not a grammatical error, she made no others). (2) Words of a certain order of difficulty (see results of Burt's Texts, p. 131) could not be pronounced correctly, e.g. 'lozenz' (lozenge), 'phiziken' (physician). But the patient never used such words spontaneously. Names, often difficult to learn even for normal adults, were not always spoken correctly, 'Mr. Broad' (Mr. Broadhurst), 'Mr. Bla-' (Mr. Black).

Adult Aphasic Patients.

No. 19 in November 1928 was almost speechless. He was never completely so during the period November 1928 to April 1929. It is only possible to summarize the main features of his record here.

(*a*) Speech was more seriously affected than silent reading, though the same symptoms were often present in the latter (p. 126). From the beginning he carried about a small notebook, compiled in his own handwriting (p. 138) but with help, containing lists of days of the week, months of the year, names of countries and places, etc., which he might want to use. Often he would take this book from his pocket and indicate, by pointing, to the name which he could not say (i.e. for the moment. See below). Latterly he has often been able to utter such words, e.g. 'Brazil', by reading them aloud from the book, when he could not say them without its help.

A clear example of this occurred during April 1929. I asked him who was going with him when he went away for a short change of air. He could not tell me, so pulled out the notebook and showed me the name written there. I asked him whether this was a friend. He wanted to explain but was unable, so fetched an atlas, opened it and pointed to the map of B——. I found that the name in the notebook was that of the friend who had accompanied him home from B—— after an illness.

(*b*) Speech was not truly 'lost'. Though he called a chair 'table', and a few minutes afterwards a table 'chair', and though by April 1929 he could rarely utter more than two or three words consecutively, when his interest was roused he used a wide variety of single words correctly and intelligently even in November 1928, e.g. 'Bowler hat', 'comparatively', 'hard business'. The greater ease with which he could make exclamations 'By Jove', 'Good', 'Great Scott', &c., might have led to the supposition that 'higher speech' (p. 84) was relatively lost. The fact seemed to be, however, that emotional stimulation revealed a large, probably normal, potential vocabulary, e.g. when in his room one day early in March 1929 I pointed to some photographs, saying, 'Is that your old regiment?' (He served throughout the War.) 'Were you in the infantry?' He repeated 'infantry' obviously puzzled (p. 126). Then I said 'Were you in the X—— Regiment?' He in his usual natural tones 'X——', then 'Gallipoli, flies, mention' (unmentionable?). I 'It must have been a terrible business'. He, tapping his abdomen, 'Dysentery, badly'. I, 'I've heard and read about the Gallipoli landings. I'm afraid there were some bad mistakes.' He 'I don't know, Churchill' indicating a copy of Mr. Winston Churchill's *World Crisis* on his book-shelves. Probably he thought I had said 'It (the campaign) was a bad mistake'. This instance is one of many which occurred. Pleasant emotions and amusement were almost as efficacious as unhappy feelings in 'freeing' his speech, and present, as well as less recent events, were discussed, though, as might be expected, the patient could talk more freely about the past.

With the exceptions discussed below, pronunciation was perfect, intonation natural, and phrasing, in the instances where words were grouped, normal. The essential difficulty,

therefore, was one of utterance under conditions which modern researches have taught us to regard as requiring the highest levels of psychological and physical function. Psychologically the act of naming objects in succession for the sake of giving information as to one's condition, or as a form of practice to improve one's speech capacity, must be regarded as a higher form of activity than the verbal expression of the wants of the moment, or of the strong emotions aroused by vivid memories of a terrible experience. The first type of act is a striving towards a more distant goal.

(*c*) Both the analytic and the synthetic aspects of the speech-language function showed disturbance. Head has described 'nominal' defects as the incapacity to fit a name to an object (p. 103).

Patient No. 19, during the period of teaching, became able to name a large number of common objects, things in his room, articles of clothing, the shops to which he went most often, with relative certainty. He began to be able to read aloud correctly short paragraphs, word by word, e.g. 'Smith went into the sitting-room. He sat down at his bureau. He wrote a letter,' &c.

Difficulty in analysis was shown in two ways. First in selecting one word from others on the page: in the early stages he often covered up all but one word which he wanted to read aloud. He also liked to do this in practising naming the hours on the clock-face—he would place a book over half of it.

Secondly, in the process of recognizing each word: he read them as whole words. Up to Dec. 1929, no attempt to help him to analyse printed words on the basis of syllables and spelling succeeded. At his first visit he could select from a number of letters of the alphabet a duplicate of one presented to him, but he could not indicate letters named aloud by me.

In reading aloud the difficulty was essentially the same as in naming objects, e.g. 'Smith' was read as 'John', 'Will' as 'Went', &c.

Before I saw him the patient had learned to copy printed or written matter with his left hand (the right being unavailable after the hemiplegia). He would write his name to order when I first saw him, but could not attempt

'dog' or 'day' to dictation, and wrote 'Wednesday' for 'Tuesday', '6' for '5', and '8' for '9'. I have verified these results since, but have not attempted to develop written language further while speech and reading are so gravely affected.

(*d*) The complexity of a situation and of the type of reaction acquired affects the efficiency of the response. This is shown by the nature of the difficulties in comprehension (p. 126) and also in the power of auditory-motor repetition. In November 1928 'eruption' was reproduced as 'ruption', and 'explosion' as 'plosion'. Now this difficulty with polysyllabic words is receding and he is becoming able to say more than one word at a time.

No. 20. With this patient the trouble appeared to take a wholly different form. The difficulty in expression lay not in 'naming' but in uttering or writing the name with correctness of detail. No. 19 had made the error of calling the fire 'bell', No. 20 called it 'higher'. No. 19 has only had difficulty in pronouncing longer and more complex words. No. 20 when I first saw him in February 1929 seemed to talk almost unintelligibly, though always with normal intonation and phrasing.

Similar errors occurred in spontaneous speech, reading and writing; e.g. in conversation, 'I come to a worst' (word); in reproducing a story, 'The straveller couldn't get a room where he was waiting' (The traveller couldn't get a room where he could stay); and 'It was winding, randing, raining badly' (this was in April 1929 when he had begun to rectify his own errors correctly); in reading aloud 'philosopher' (philosophy), 'steeple-shase' (steeple-chase), 'house' (horse), 'moker-car' (motor-car), 'West-ned' (West End), &c.; in writing 'Thank yot' (Thank you), 'Manc H', Hanchac, Hanehaugh (Manchester), these written examples being obtained at his first visit.

Perseveration was very marked with this patient—he read 'thrush, thrusting' as 'thrush, thrushing', and after naming correctly 'spoon' being faced with an illustration of a setting sun he said 'S', 'S, double-O-N, soon'. A moment afterwards he named a picture of snow as 'soon'. 'Golf-clubs' were 'glof-glubs'. Just as No. 19 in reading had difficulty in isolating one word on a page, so No. 20 tended to introduce

later syllables at the beginning of a word, e.g. 'prous' for 'pronounce' or even to confuse a later with an earlier word in a sentence.

No. 20 has shown remarkable improvement during the period of teaching. In December 1929 he can talk fluently about any subject, though hesitations and errors of the types just illustrated occur occasionally. Earlier in the chapter it was mentioned that this patient has learned to read books as well as newspapers and correspondence. He can also write or type-write an original letter with at most two or three slight mistakes in spelling. He has been most helped by re-education in reading. He has become able to refer mentally to the spelling of words and to check his speech by it.

An analysis of the psychological phenomena exhibited by the patients No. 19 and 20 leads to the following conclusions:

(*a*) According to the teachings of Marie and Moutier these two patients suffered from the syndrome, 'Anarthrie pure + aphasie'. But the statement that anarthria involves 'incoordination of the speech organs' hardly covers the perfect articulation of most words by No. 19.

(*b*) Under Von Monakow's scheme of 'clinical classification' both patients might be described as suffering from aphasia of motor and expressive type. If so, these patients suggest that von Monakow may have underestimated the defects of comprehension in such cases. In neither instance was intonation monotonous as he described, and perseveration was much more marked in No. 20 than in No. 19. No. 20 uttered long sentences, No. 19 groups of two or three words at most. It seems probable that the period of diaschisis is past in both cases.

(*c*) Head's classification of aphasic defects is applicable in so far that No. 19's trouble was outstandingly 'nominal' and No. 20's 'verbal'. But No. 20's rapid utterance, normal intonation and rhythm in speech are inconsistent with Head's description of 'verbal' defects.

Using these two adult aphasic patients as a link through personal experience with the data of past investigations, it is now possible to form conclusions in regard to the aetiology of the defects presented by the 'aphasic' children.

SUMMARY OF CONCLUSIONS

1. *Hearing Capacity of 'Aphasic' Children.*

As a result of using original tests, six (Nos. 1–6) out of ten 'aphasic' patients manifesting either absence of or gross defects of speech and language in childhood, were proved to suffer from a marked, binaural, and evenly progressive lack of hearing, for ascending orders of frequencies in sound above 256 v.d. This deficiency was associated with normal or relatively normal hearing for sounds of frequency below 256 v.d. The other four patients (Nos. 7–10) had normal hearing for all the frequencies tested. One of these four, No. 7, had been admitted to a school for the deaf as suffering from total deafness of congenital origin. The aetiology of her trouble was unknown before she was examined in the course of this research.

2. *High-Frequency Deafness.*

It was proved that the lack of auditory acuity for the higher frequencies had abolished the characteristic differences of the sounds of speech and most noises for patients Nos. 1–6. This explained their indifference to sound before teaching by special methods. The evidence was as follows:

(*a*) Defects in auditory acuity were considered in relation to positive analyses of the sounds of human speech.

(*b*) The six high-frequency deaf children, through teaching, all learned to recognize and understand speech by ear alone. A series of tests was evolved which confirmed the conclusion that their ability, thus acquired, to comprehend the spoken word depends on their nearness to the source of sound and the intensity of the characteristic partial tones.

3. *Minimum Hearing essential to Normal Speech and Language Development.*

An analysis of the results of testing all of the 10 patients and 12 normal children, 70 deaf children (+ 1 with normal hearing who had lost speech through shock), 2 normal adults, and 5 other deaf patients—a total of 100 individuals, and a comparison of these results with those obtained by Zuehl,

Fletcher, and other workers, led to the conclusion that the maximum lack of auditory acuity, of incidence earlier than 5 years of age, consistent with normal speech and language development, was represented approximately on our audiometer by the intensity steps 50 for frequencies 64–256 v.d. (fundamental, laryngeal tones) and 45 for the range of vowel and consonant characteristics, 512–8,192 v.d.

4. *High-Frequency Deaf Children Not Aphasic.*

The speech and language development of the six high-frequency deaf patients, Nos. 1–6, passed through the same stages and showed the same departures from normal adult speech as those of the normal child. These six patients showed no traces of the defects characterizing aphasia. Before coming to this conclusion a careful study was made of the data obtained from the principal investigations into aphasic conditions occurring among both adults and children. Two adult aphasic patients were studied and taught for 17 and 12 months respectively.

5. *Aetiology of High-Frequency Deafness.*

The constant decrease in auditory acuity for ascending orders of frequency of sound found in the six patients, considered in conjunction with the results obtained with patients suffering from inner ear trouble and acoustic nerve defects by Bunch with an Iowa audiometer, Nemzek and Hays with a 2A Western Electric audiometer, and the results of the aural examination of the patients themselves by Dr. H. C. Smith, suggests that the defect may be in the cochlea, along the auditory path, or possibly even due to late myelination of nerve fibres associated with hearing.

6. *Linguistic Retardation.*

Four patients (Nos. 7–10) were found to have normal hearing for sounds of all the frequencies tested. Yet they exhibited, in childhood, defects which in the past would have been described as aphasic. The terms 'congenital aphasia', 'word-deafness', and 'word-blindness' have been used in such cases by various investigators (pp. 105–10).

From the data obtained during this research only one conclusion seems possible with regard to these four patients

(Nos. 7–10), namely that aphasia is no more a correct description of their forms of trouble than it is of high-frequency deafness.

It is impossible to find any similarity in essential features, between the failure to achieve progress normally in speech and language development, shown by patients Nos. 7–10, and the disorders resulting from the breakdown of the central mechanism for speech, as the effect of either disease or injury in the brain, after full normal development has once been attained.

The patients Nos. 7–10 cannot be classified under any of the types of aphasia described by Head, Pick, Von Monakow, or earlier workers.

The features of aphasia in adults are variability in response to the words of others and in the spontaneous use of words, and dissociation. When the adult aphasic misunderstands or misuses words it is because only part or parts of his aggregate of speech habits are reacting to part of the external environment. Complete adaptation is not being attained. There is often a strong element of conflict in his mistakes. The details of this may be traced out by taking actual examples, for instance, those recorded in this book, and trying to calculate what factors due to the patient's past history, the present or the immediately preceding situation, attained undue or insufficient force.

The patient's own efforts to speak or to write are an important feature of the situation. The first movement required to produce a word may lead him more easily to a movement belonging to another word than to its correct successor. In adult aphasia there is also a degree of lowered excitability of the speech language function which appears to be in proportion to the severity of the damage to the brain.

By contrast, the speech and language of the children (Nos. 7–10) were characterized by uniform, predictable, and as it were fully focused reactions. Another marked feature was that the mistakes of these children are observable among normal children at certain stages of development. This is the reason why the description 'linguistically retarded' has been adopted in this book in place of 'congenitally aphasic'. The aetiology of linguistic retardation, in the absence of any

evidence of hereditary tendencies, appears to be some pathological factor, possibly in some cases a period of lowered general vitality, retarding or arresting speech and language at a specific stage.

That the speech and language function, the most distinctive characteristic of the human species, is more susceptible than any other to pathological conditions affecting the brain, is evident from the facts of adult aphasia. That the individual having missed or only partially attained the steps of language development, in their natural sequence and in the first years of life, can never attain complete command of language by the simple untutored process of hearing and imitation, is a fact made familiar by the methods employed to teach foreign languages.

Each of the linguistically retarded patients described in this book showed symptoms explicable on these grounds.

No. 7, at the age of 4 yrs. 9 mths., was still in the prelinguistic stage. To-day, within her limits of attainment, her speech and language habits are uniformly reliable.

No. 8 at 7 yrs. had incompletely emerged from the 'one-word sentence' period, but within his limits his speech and language were reliable. To-day (December 1929), after teaching, he is able to talk almost like a normal child.

No. 9, at 11 yrs., showed by his speech that there had been a retarding factor in the early period of babbling and imitation of vowel and consonant sounds. With teaching he was able to learn to speak correctly.

No. 10 was the least retarded of these patients; 'word-blindness' was only the most obvious feature of her trouble, as Fildes, Burt, and Hincks had noted in other instances. The defect in verbal imitation was not discoverable except under conditions of unaccustomed difficulty, especially when longer words and literary phraseology were introduced. Superficially her mistakes in reading and writing resemble those of an adult aphasic.

The evidence that they were of a different nature was first the character of the defect in verbal imitation, and secondly the reliable progress in reading and writing, again within limits, made after teaching.

A detailed comparison of these mistakes with those of adult

aphasics, such as patients Nos. 19 or 20, or those described by other workers, reveals a complete contrast. So far as No. 10 has learned to use words, she can use them with certainty and accuracy. The errors occur in efforts which take her beyond her standard of language development, or when strange and complex conditions unduly tax her speech and language capacity. The typical features of adult aphasia, dissociation, conflict, and in so many instances strongly marked perseveration, are completely absent.

The earlier mistakes in oral repetition and in writing are more accurately comparable to those of the half-educated individual than to those of the adult aphasic.

The final conclusion is that there are fundamental distinctions in symptoms and in aetiology between failures in natural speech development in childhood and the breakdown of the perfected processes in later life.

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APPENDIX

AURAL EXAMINATION OF HIGH-FREQUENCY DEAF PATIENTS Nos. 1–6

on March 19th and 22nd, 1929, by H. C. Smith, M.B., Ch.B. (Vict.)

No. 1: The membranes were normal in appearance. They were rather delicate, thin, and the ossicles pale and slightly undersized.

There was no evidence of disease, of any past or present pathological process in the conductive apparatus, and the throat and nasal pharynx were perfectly healthy.

No. 2: The appearance of the memb. tympani were very similar to those in No. 1, in that there were no signs of disease or recovery from such. Questionable tonsils, and a slight catarrhal condition of the nasal pharynx may be a factor intermittently increasing deafness, but quite unimportant relative to the main loss of hearing and nothing to do with the causation.

The membranes show opacity to a slight degree, but are more durable than No. 1's, and a little less mobile.

Conclusions in regard to patients Nos. 1 and 2.

I am inclined to think that these two children can be placed in the same category of deafness, viz. born deaf: possibly a pathological process acquired *in utero*, and affecting the lower portion of the cochlear apparatus. The conductive apparatus is normal except for slight impairment of mobility of the membranes and ossicles. The preservation of lower tone limits and intensities, with loss in the upper tones, points to affection of the lower part of the cochlea, and is consistent with undamaged conductive apparatus.

No. 3: Right tympanic membrane opaque, much retracted, has appearance of post-suppurative condition of middle ear. Left: appearance of totally disorganized middle ear, tympanic membrane absent, and enlarged deformed malleus, displaced.
Probably post-natal suppuration trouble.

No. 4: Meatus R. and L. plugged with hard cerumen.

No. 5: Tympanic membranes intact. No evidence of perforation or pathological process. Impaired mobility, sclerosed appearance of malleus. Probably inherent congenital condition.

No. 6: Right tympanic membrane slightly retracted, intact and glistening and normal shadow. No evidence of perforation. Left: similar appearance but drum more opaque and more retracted. (Tonsils enlarged, not healthy.) Trouble may be of congenital origin.

Additional report on two other patients, Nos. 202 and 221. (Deaf Children. Table C, p. 42.)

No. 202: Opaque membranes, partly hidden by soft cerumen and

debris. Probably post-suppuration condition middle and inner ears. Again possibly following measles.

No. 221: Right: pale atrophied malleus, retracted glistening membrane, with evidence of an old healed perforation. Left: slight retraction and opacity of membrane. Signs of post-nasal catarrh; a post-nasal suppuration probably caused a suppurative middle-ear condition with onset of deafness. Possibly measles, with labyrinthine trouble.

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